"COMPARISON OF SPHENOPALATINE AND GREATER PALATINE BLOCK IN MINIMISING BLEEDING DURING ENDOSCOPIC NASAL SURGERY"

By

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DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF

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In partial fulfilment of the requirements for the degree of

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IN
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Under the Guidance of

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DR AISHWARYA RAJ PILLAI D

LIST OF ABBREVIATIONS

1.	FESS	Functional Endoscopic Sinus Surgery
2.	PPF	Pterygopalatine Fossa
3.	CRS	Chronic rhinosinusitis
4.	ARS	Acute rhinosinusitis
5.	CRSwNP	Chronic rhinosinusitis with nasal polyps
6.	CRSsNP	CRS without nasal polyps
7.	ITH	Inferior turbinate hypertrophy
8.	DNS	Deviated nasal septum
9.	NP	Nasal polyposis
10.	RAST	Radioallergosorbent testing
11.	COX	Cyclooxygenase
12.	IL	Interleukin
13.	NSAID	Non-steroidal anti-inflammatory drugs
14.	HLA	Human leukocyte antigen
15.	AR	Allergic rhinitis
16.	IPPV	Intermittent positive pressure ventilation
17.	MAP	Mean arterial pressure

ABSTRACT

Background: Endoscopic nasal and sinus surgery is one of the most common surgeries in otorhinolaryngology. The complex anatomy with its unique variations and its vicinity to important structures require the surgeon to precisely identify structures in a clear bloodless field. Even with recent advances in technology, controlling the bleeding during endoscopic sinus surgery remains a challenge

Aims and objectives:

1)To evaluate haemostasis following sphenopalatine block in endonasal surgery.

2)To evaluate haemostasis following greater palatine block in endonasal surgery.

3)To compare the effective haemostasis between greater palatine and sphenopalatine blocks.

Materials and methods: This study was done on patients undergoing endoscopic nasal and sinus surgery from December 2017 to June 2019. Patients were selected based on the inclusion and exclusion criteria. Each patient received greater palatine block on one side and sphenopalatine block on another side with 2 ml of Inj 2% Lignocaine and 1:100000 adrenaline and intra-operatively surgical field assessment was done every 15 minutes to assess the bleeding and was graded using Boezart and Van der Merve scale

Results: Maximum incidence of disease was seen in the 3rd and 4th decade of life. B/L sinonasal polyposis was the most common diagnosis 50%, followed by Chronic rhinosinusitis 26% and 24% had allergic rhinitis. Mean Blood loss grade for sphenopalatine block was 2.48± 0.26 and for greater palatine block was 2.42 ± 0.30 which showed there was no significance difference between the 2 blocks with regard to haemostasis. No significant correlation was found between the sphenopalatine and

greater palatine blocks and intraoperative heart rate, SBP, DBP and MAP. No complications were encountered during the procedure.

Conclusion: Both greater palatine block and sphenopalatine block provided fairly good haemostasis facilitating good visualization of the surgical field and requiring occasional suctioning. There was no significant difference in haemostasis between sphenopalatine block and greater palatine block**.**

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INTRODUCTION

The surgical field in endoscopic nasal and paranasal surgeries is very narrow and is surrounded by vital structures. Even with recent advances in technology, controlling the bleeding during endoscopic sinus surgery remains a challenge. Even a small amount of bleeding during surgery can soil the tip of the endoscope and obscure the field. Repeated soiling of the tip of endoscope prolongs the procedure. Operating under such compromised conditions and in addition the narrow and delicate field increases the risk of injury to the adjacent vital structures. Hence an optimal bloodless field is crucial for the surgeon. [1]

Various methods have been used to secure a dry operating field, among them are: Fowler's position, hypotensive agents like beta-adrenergic blockade, and preoperative steroids, topical vasoconstrictors. However, even with all these techniques, excessive bleeding still remains a challenge during endoscopic nasal surgery.[2,3]

Maxillary artery is major source of blood supply to the nose. Therefore vasoconstriction of this artery or its branches can significantly minimise bleeding during procedure.[1] This can be achieved by sphenopalatine block or greater palatine block.

There have been only few studies comparing the effective haemostasis of sphenopalatine and greater palatine block in endoscopic nasal surgeries. Therefore, this study was done to document haemostasis with each one of these blocks and compare the haemostasis between the two.

OBJECTIVES OF THE STUDY

1)To evaluate haemostasis following sphenopalatine block in endonasal surgery.

2)To evaluate haemostasis following greater palatine block in endonasal surgery.

3)To compare the effective haemostasis between greater palatine and sphenopalatine blocks.

REVIEW OF LITERATURE

Throughout the history of medicine, numerous attempts have been made to illuminate and examine the inner side of various hollow cavities in our body. The interior of nose and paranasal sinuses, with their narrow passage and fissures, bony walls place heavy demands on the design of instrumentation to be used for this purpose. This sowed the seed for development of nasal endoscopy.[4]

. Philip Bozzini in 1806 published an article describing the first "Light conductor, or description of a simple device and its use for illumination of the internal cavities and spaces of living animal body". In 1915 Killian published a review of the "History of endoscopy from the earliest times to Bozzini "in which he recorded all the attempts to view the upper airways prior to beginning of 19th century. [4]

Bozzini mentioned in 1806 that he was able to see some areas behind soft palate with the aid of his light conductor. In 1838 Baumes presented to medical society in Lyons a mirror the size of two franc pieces that could be used for the examination of choanae and the larynx.[4]

In 1859 in Vienna, Czermak developed a technique similar to laryngoscopy of Turck, which allowed him to view the nasopharynx, the choanae and the posterior aspect of nose with the aid of small mirror. He called this procedure "Rhinoscopy"[5].

The second stage of cystoscopy began with the development of cystoscope by Nitz-Leiter in 1879. A year later Zauful modified the instrument for examining eustachian tube orifice. In 1902, Hirschmann and Valentin followed shortly by Reichert, in 1903 were able to introduce a modified cystoscope into the maxillary sinus through

an enlarged dental alveolus.[4]

During 1951-1956 Hopkins made fundamental improvements in the optics of endoscopy. These included a light source that was separate from the instrument, an excellent resolution with high contrast, a large vision in spite of the small diameter of the endoscopes and perfect fidelity of colour. The Hopkins rod rigid nasal endoscopes made it possible to examine in detail the clefts and recesses of the nose. The ability to enter middle meatus of the nose enabled the inspection of anterior ethmoid sinuses and key area of infectious paranasal sinus diseases. Today nasal endoscopic examination in combination with tomography allows the identification of small circumscribed changes in paranasal sinuses. These small changes are usually carry pathophysiological significance.[4]

A systemic endoscopic diagnostic approach to lateral wall of nose was first to developed and established by Messerklinger. His studies beginning in 1950 demonstrated that in most cases the frontal and maxillary sinuses are involved indirectly by primary disease that originates in narrow spaces of the lateral wall of nose and the anterior ethmoid. This discovery with the aid of rigid endoscopes and computed tomography of sinuses resulted in the improvement of endoscopic diagnostic technique that focused on changes on the lateral wall of nose and identified and isolated changes. Messerklinger observed that the eradication of only the primary anterior ethmoid disease resulted in resolving of massive mucosal pathology in the adjacent large paranasal sinuses within a few weeks.[4]

Endoscopic nasal surgery is one of the most common surgical procedures done under direct visualization; the main advantage being less invasive when compared to conventional procedures. Due to the complex anatomy and proximity of vital structures such as intracranial and infraorbital compartments endoscopic nasal

surgery can be difficult.

The narrow confines of the surgical field can become easily obscured even by small amounts of bleeding. Hence to get optimal visualization, decrease operation time, reduce complications and improve outcomes, it is optimal bloodless field is crucial for the surgeon.

Various techniques and methods have been used to maintain a dry operating field in endonasal surgeries among them are topical vasoconstrictors, hypotensive anaesthesia, preoperative steroids.[5]

Local injection with local anaesthetic and adrenaline to the pterygopalatine fossa either by transoral greater palatine fossa or the transnasal sphenopalatine foramen region have shown to cause better haemostasis due to vasoconstriction of the vessels of the lateral wall of the nose caused by adrenaline, due to the direct tamponade effect of the injection on the sphenopalatine artery and a parasympathetic block allowing unopposed sympathetic activity.[2,6]

Wormald states that locating the sphenopalatine foramen is difficult and hence the injection through the greater palatine fossa which can be easily located just anterior to the posterior edge of hard palate opposite the second molar is more reliable.[2]

Wormald et al have reported significant decrease in estimated blood loss during FESS with greater palatine block. [1]

A study done in Kolar region published in 2017 found that haemostasis was better with pterygopalatine block given via greater palatine foramen.[7]

Cho et al have shown that sphenopalatine block improves surgical bloodless field and also improves post-operative analgesia.[8]

The drawbacks of these blocks are

These are blind procedures; hence to be more accurate imaging guidance can be used

Injury to sphenopalatine artery

If excess is injected, then it can cause orbital vessel vasospasm which can cause blindness

These blocks can in addition be used to give post-operative analgesia and headache.[9,10,11,12]

ANATOMY OF NOSE AND PARANASAL SINUSES

EMBRYOLOGY OF NOSE

Nasal cavity is first recognized in the 4th week as an olfactory or nasal placode. The placode sinks to form the olfactory pit. This then deepens to form the nasal sac. The maxillary process of the 1st arch grows anteriorly and medially to fuse with nasal fold and fronto-nasal process. This closes off the nasal pits to form the primitive nasal cavity.[13]

Initially mouth and primitive nasal cavity are separated by bucconasal membrane. This thins as nasal sac extends posteriorly and eventually breaks down to form primitive choana. The floor anterior to the choana is formed from mesenchymal extensions of medial nasal folds to produce premaxilla which gives rise the upper lip, medial crus of lower lateral cartilages.[13]

The maxillary process also grows ventrally form dorsal end of mandibular process to join the lateral nasal process around the nasomaxillary groove. The ectoderm in this region canalizes to from nasolacrimal duct. The lateral nasal folds form the nasal bones, upper lateral cartilages, and lateral crus of lower lateral cartilage. Palate begins to form anteriorly with the fusion of the maxillary and fronto nasal process. Nasal septum is formed from the midline ridge developing from the posterior edge of fronto nasal process in roof of oral cavity and extends posterior to the opening of Rakthe's pouch.[13]

The palatal process from lateral maxillary mesoderm grows medially towards septum and towards each other. Fusion is complete, except a midline dehiscence which forms site of future incisive canal. It separates the nasal cavity and nasopharynx from oral cavity as they also form soft palate and uvula.[13]

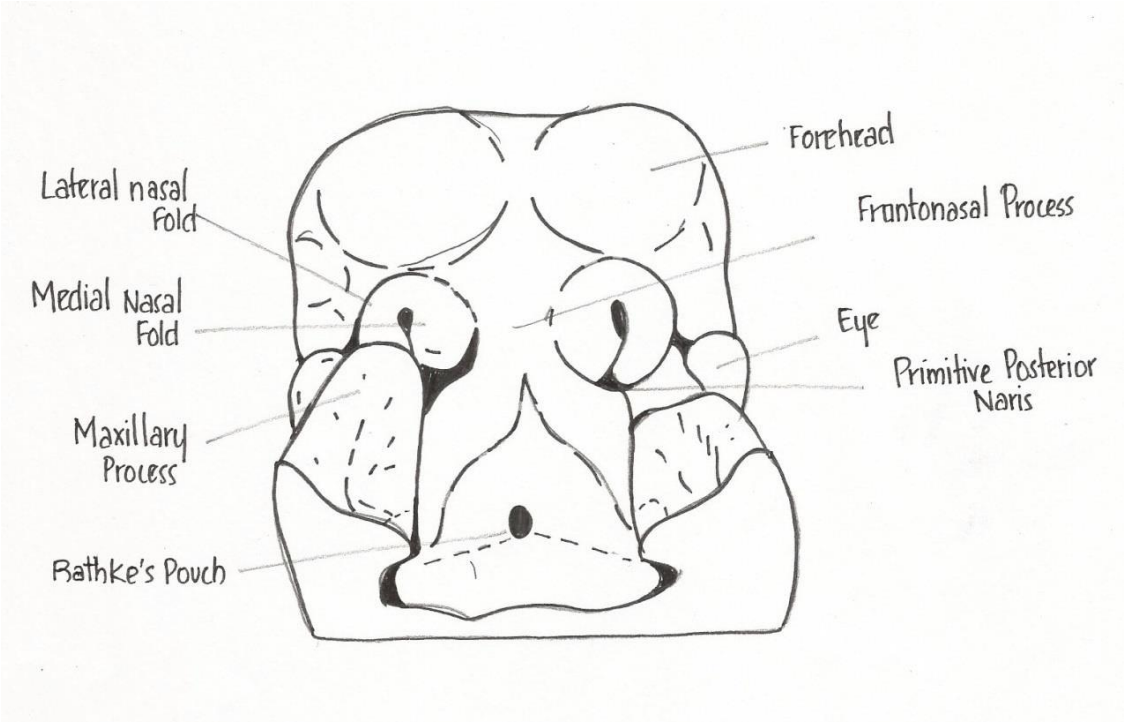


Figure 1: Development of Nose

ANATOMY OF NOSE

EXTERNAL NOSE

The external nose with its root above and base directed downwards is shaped like a pyramid. The two nostrils or anterior nares perforate the base, separated by median septum. Each side of external nose ends in a rounded eminence, the ala nasi, which forms the outer boundary of the nostril. The nasal bones form the bridge, and each is united above with frontal bone and laterally to frontal process of maxilla. Two paired cartilages, the lower and upper lateral cartilage and one unpaired cartilage, the septal cartilage, complete the external framework.[1]

The external nose skin receives its sensory supply from the two upper divisions of the trigeminal nerve; ophthalmic and maxillary. Blood supply is from maxillary and

ophthalmic arteries. The anterior facial vein and ophthalmic vein forms the venous drainage, lymphatics drain to the submandibular and pre-auricular lymph nodes.

NASAL CAVITY

Each nasal cavity is divided into three parts i.e., nasal vestibule, olfactory region and respiratory region.

Nasal vestibule is the most anterior and it extends from the nostril antero-inferiorly to the nasal valve postero-superiorly. The nasal valve is situated between the caudal end of the upper alar cartilage laterally and the septum medially. The area of demarcation is limen nasi, with skin containing hair follicles, sebaceous and sweat glands. This area is of importance since it is here that nasal cavity is the narrowest, limited to a triangular shape of only 0.3 square cm on each side.

The olfactory region is confined to the roof of the nasal cavity and the superior turbinate representing an area of 10 square cm. The remainder area of nasal cavity constitutes the respiratory region and its surface may reach 120 square cm.[13]

NASAL SEPTUM

The septum of the nose is formed by the perpendicular plate of ethmoid, the vomer, nasal crest of maxillary and palatine bones and septal cartilage.

The main arterial supply of nasal septum is from the septal branch of sphenopalatine artery. The antero- inferior part of the septum or Little's area is where the septal branches of sphenopalatine, greater palatine, anterior ethmoidal and superior labial artery anastomose. Venous drainage occurs to pterygoid plexus. The anterior septum drains into submandibular nodes while posterior drains into the retropharyngeal and

anterior deep cervical nodes. The nerve supply is by nasopalatine nerve posteriorly and by the anterior ethmoid branch of nasociliary nerve and antero-superior alveolar nerve anteriorly.

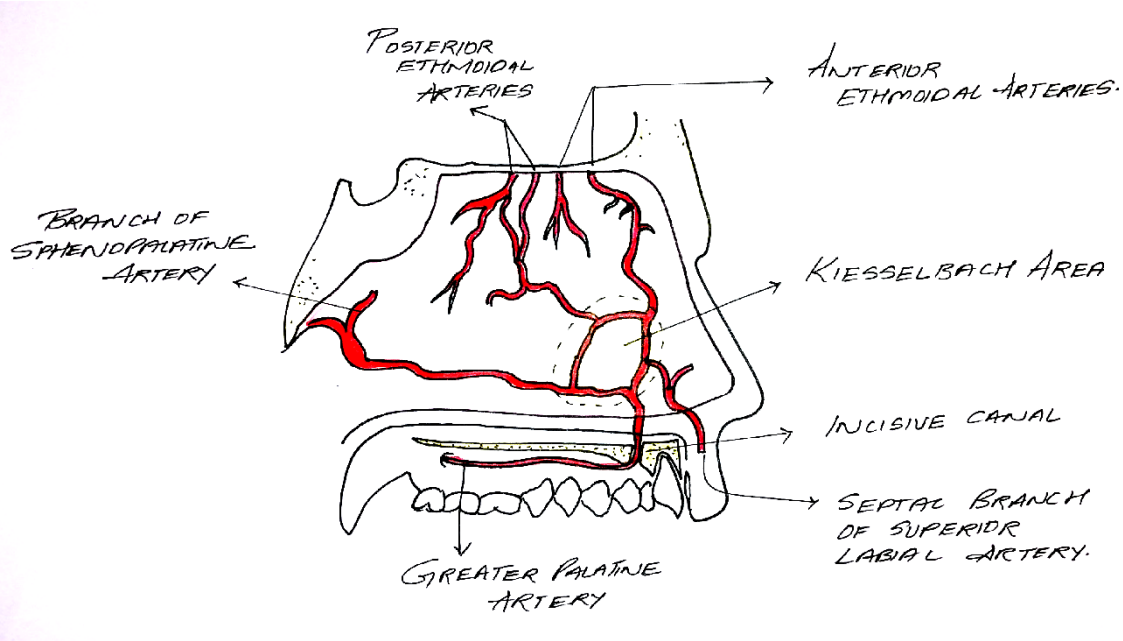


Figure 2: Blood supply of the septum

LATERAL WALL

Lateral wall has a characteristic complex structure as a result of scrolls and projections present on it, which are convoluted to form turbinates. These projections are the superior, middle, and inferior turbinates and in 10% of patients the supreme turbinate maybe present. The inferior turbinate is a separate bone, whereas middle and superior turbinates are projection from ethmoid. The superior, middle and inferior meati are found below and lateral to roof of each of respective turbinates.[6] The posterior third of the lateral wall occupied by the superior meatus, the posterior two thirds is occupied by middle meatus and the inferior meatus runs the whole length of the lateral wall. Olfactory cleft is the space between the nasal septum and

the middle turbinate. Posterosuperior to superior concha the space is known as sphenoethmoidal recess in which sphenoid sinus opens. The air cells of posterior ethmoid open into the superior meatus. Into the middle meatus the frontal, the anterior and the middle ethmoidal air cells and the maxillary sinus open. The nasolacrimal duct opens in the anterosuperior portion of the inferior meatus at the point where the inferior concha lies in contact with lateral wall of the nasal cavity.[14]

NASAL MUCOUS MEMBRANE

Nasal mucous membrane consists of dense connective tissue. The mucous membrane is predominantly respiratory epithelium with a small area of olfactory epithelium superiorly adjacent to the cribriform plate. Respiratory epithelium is made of ciliated and non-ciliated pseudo stratified columnar cells, basal pluripotent stem cells, and goblet cells. Seromucinous glands in the sub mucosa are more important in mucous production in nasal cavity than goblet cells which are numerous in sinuses.[13]

NERVE SUPPLY OF NOSE

Nerve supply to nose is extremely rich and includes the general sensory, parasympathetic and sympathetic innervations. The main sensory supply comes from maxillary division of the trigeminal nerve from its pterygopalatine branches (nasopalatine, greater palatine, short palatine nerve). The lateral and medial internal nasal branches of the anterior ethmoidal nerve supply the anterior part of the nasal cavity. The anterior end of the inferior turbinate and floor are supplied by branches of anterior superior alveolar nerve. The secretory nerve fibers arise from the

sympathetic and parasympathetic systems. Sympathetic fibers originate through superior cervical ganglion via the sympathetic plexus of internal carotid artery and merge with the parasympathetic fibers in the pterygoid canal. These contribute to the nose vasculature via the greater superficial petrosal nerve to form nerve to pterygoid canal. Parasympathetic fibers derived from the superior salivary nucleus in the medulla via the nervus intermedius to form the vidian nerve and reach sphenopalatine ganglion, where they relay before entering the nasal cavity and supply blood vessels of the nose causing vasodilatation. The special sensory olfactory nerves are distributed in a network in the mucosa in the upper third of nasal septum, roof and corresponding part of the lateral wall of nasal cavity.[15]

BLOOD SUPPLY

Nasal cavity receives its blood supply from branches of both internal and external carotid arteries. The anterior and posterior ethmoidal arteries which are branches of sphenopalatine and greater palatine, both are branches of maxillary artery and superior labial branch of facial artery supply anterosuperior quadrant of nose. The demarcation between the two carotid system is at the level of the middle turbinate.

Venous drainage is by formation of cavernous plexus beneath mucous membrane and drains through the sphenopalatine and facial veins. Woodruff's plexus is a venous plexus in the posterior part of the inferior meatus which is the most common site for posterior epistaxis. Lymphatic drainage from the anterior part of nose is to the submandibular nodes and to the superior nodes of deep cervical chain. The

posterior part drains into the middle and deep cervical chain.

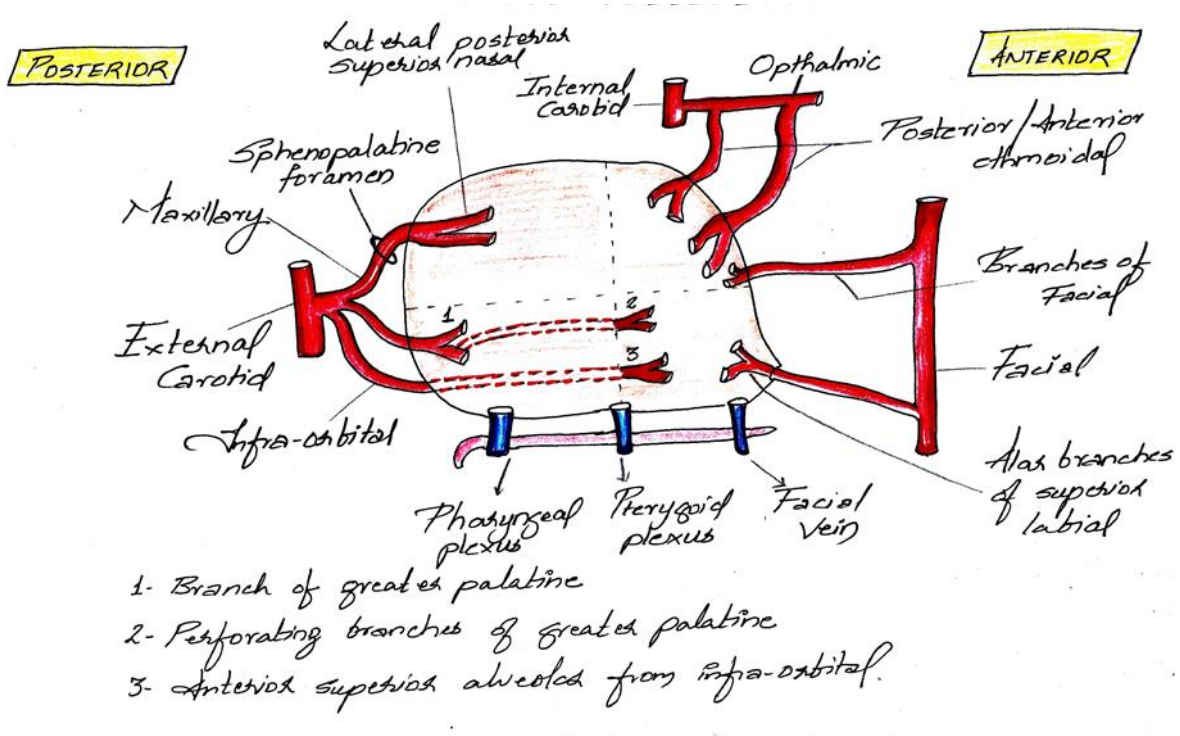


Figure 3: Blood supply of Lateral wall of Nose

OSTEOMEATAL COMPLEX

Neumann coined this word to describe the region comprising middle meatus with the anterior air cells. This is the most crucial area for normal paranasal sinus functioning and any pathology in this area will disrupt the physiology and leads to sinus dysfunction. In the middle meatus there are several important structures. Anteriorly the first landmark is a hook shaped bone called the uncinate process. Hiatus semilunaris is a groove posterior to the uncinate which leads to the ethmoidal infundibulum. A bulge posterior to the hiatus is called the ethmoidal bulla, which is a part of anterior ethmoidal group of cells. The frontal sinus opens into the superior most aspect of the ethmoidal infundibulum called the frontonasal recess, while the anterior ethmoidal cells open into the infundibulum. The ostium of maxillary sinus

opens posteroinferiorly into the infundibulum.[13]

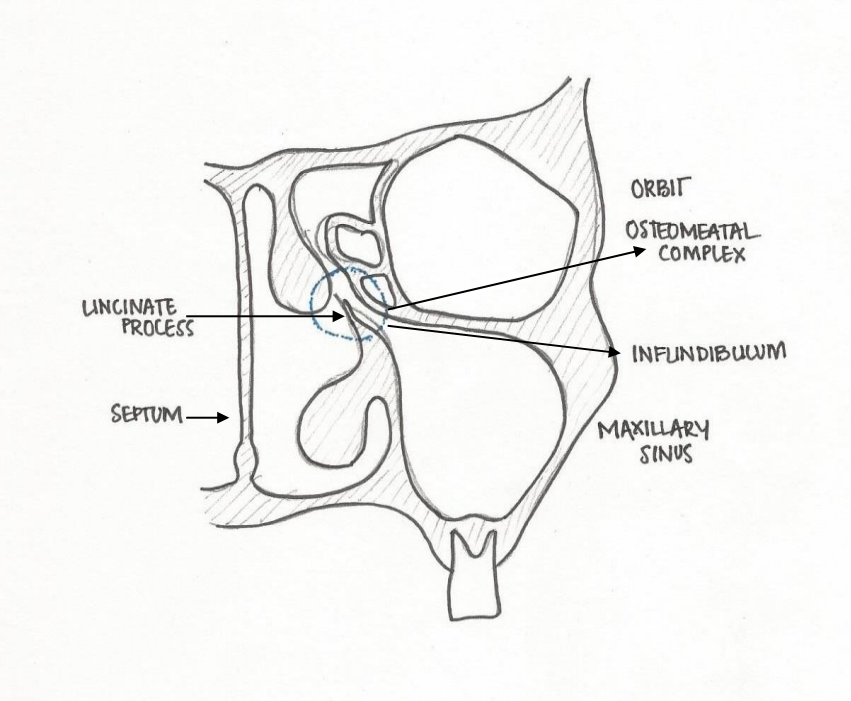


Figure 4: Osteomeatal complex

PHYSIOLOGY OF NOSE

The principal functions of the nose include

I. Olfaction

II. Airway

III. Air conditioning

IV. Airway resistance

V. Resonance

VI. Reflex functions

PARANASAL SINUSES

EMBRYOLOGY OF PNS

MAXILLARY SINUS

The maxillary sinus is the first sinus to appear at 7-10 weeks as a shallow groove expanding from the primitive ethmoidal infundibulum into the mass of the maxilla. It continues to grow during childhood at an estimated annual rate of 2 mm vertically and 3 mm anteroposteriorly and in particular with development of middle third of face as the dentition erupts. It attains its final size by 17-18 years.

The maxillary sinus in an adult pyramid shaped with the base formed by the medial wall of the maxillary sinus and apex of pyramid towards the zygomatic recess.[13]

ETHMOID SINUS

At 9-10 week of gestation, six major furrows appear on the lateral wall of nose. The furrows are separated by ridges which have an ascending portion called ramus ascendens and a posteroinferior portion called ramus descendens. The inferior turbinate is also called the maxilla-turbinal and is an individual bone. The first ethmoturbinal regress and the descending portion gives rise to the uncinate process, the ascending process forms the agger nasi. The first furrow gives rise to the infundibulum and the frontal recess. Middle turbinate is formed from the second ethmoturbinal, superior from the third. The fourth and fifth ethmoturbinals regress during development. [13,14]

SPHENOID SINUS

The sphenoid sinus is recognizable at around the third intrauterine month as an

evagination from the sphenoethmoidal recess and again a small cavity is found at birth. At the third year of life, pneumatization of the sphenoid bone progresses and at age seven has frequently reach the floor of sella.[13,14]

FRONTAL SINUS

The frontal sinus is the most variable in size and shape and maybe regarded embryologically as an anterior ethmoidal cell. From the most anterior and superior segment of the anterior ethmoid complex, the frontal bone is gradually pneumatized, resulting in frontal sinuses of variable size. At birth the frontal sinuses are small and on x-rays cannot be differentiated from other anterior ethmoidal cells.

ANATOMY OF PARANASAL SINUSES

Paranasal sinuses are arranged in pairs and include two groups anterior and posterior. The anterior group includes maxillary sinus, frontal sinus and anterior ethmoidal sinuses. The posterior group comprises of posterior ethmoidal and sphenoidal sinuses.[13]

MAXILLARY SINUS

It is present since birth but attains its maximum size around 15 to 17 years of age. The roof is by floor of orbit, floor by roots of canine alveolus. Posteriorly, it is related to infratemporal and pterygopalatine fossa (PPF), anterolateral walls are superficial and just deep to soft tissues of face, medial wall formed by nasal cavity. The maxillary ostium present at the upper portion of sinus, opens into the middle meatus. In some, an accessory ostium maybe present posterior to the main ostium.[13,14]

FRONTAL SINUS

At birth it is rudimentary, being represented by a small upward prolongation from

anterior end of middle meatus, the nasofrontal duct. It is bound anteriorly and posteriorly by the inner and outer table of frontal bone, floor by roof of orbital cavity, medially by septum between the two frontal sinuses. The ostium of frontal sinus is situated in its floor, drains into middle meatus.

ETHMOIDAL SINUS

It is present at birth and they vary in number, size and shape. They are grouped into anterior and posterior, depending on whether they communicate with middle or superior meatus. They are bound medially by upper half of nasal cavity, laterally by orbit, anteriorly by frontal process of maxilla and posteriorly by sphenoid bone.[14]

SPHENOID SINUS

It is present at birth. The roof is related to frontal lobe, olfactory tract, optic chiasma and pituitary gland and lateral wall is related to internal carotid artery, optic nerve and the cavernous sinus. The floor is related to pterygoid canal, medial wall is between the two sphenoid sinus. The sphenoid ostium is situated high up in the cavity of sinus.[13,14]

PHYSIOLOGY OF PARANASAL SINUSES

Air conditioning: They serve as supplementary chambers for conditioning the inspired air by heating and moistening.[13,16]

Vocal response: They act as resonating chambers and add to quality of voice.

Thermal insulators: They safeguard the structures in orbit and cranial fossa from intra temporal variations.

Balance of head: It reduces the weight of the bones of face, thereby aiding in balance of head.[13,16]

MUCOCILLIARY CLEARANCE

Drainage and ventilation are two most important factors in maintenance of normal physiology of paranasal sinuses. It depends on the quantity of mucus produced, composition of mucus, effectiveness of ciliary beat, mucosal resorption, condition of ostia and ethmoidal clefts.[16]

The mucus film has two layers: an inner serous layer, called the sol phase, in which cilia beat and an outermost viscous layer, the gel phase, which is transported by the ciliary beat. This functions like a conveyor belt. Normal nasal mucus exists at a pH range of 7.5 to 7.6.[16]

In maxillary sinus secretion transport starts from the floor of sinus in a stellate pattern. The mucus from anterior, medial, posterior, lateral wall and roof of sinus converge at the natural ostium. This is finally drained into middle meatus. Frontal sinus has active inward transportation of mucus. Due to whorled pattern of cilia, mucus is circulated again and again. Finally, mucus from frontal sinus drains into frontal recess. The anterior ethmoidal cells drain into middle meatus and into sphenoethmoidal recess drain the posterior ethmoidal cells. In the sphenoidal cells, mucus undergoes a spiral transport and drains into sphenoethmoidal cells. All these secretions finally go to the lateral nasal wall and from there into the nasopharynx.

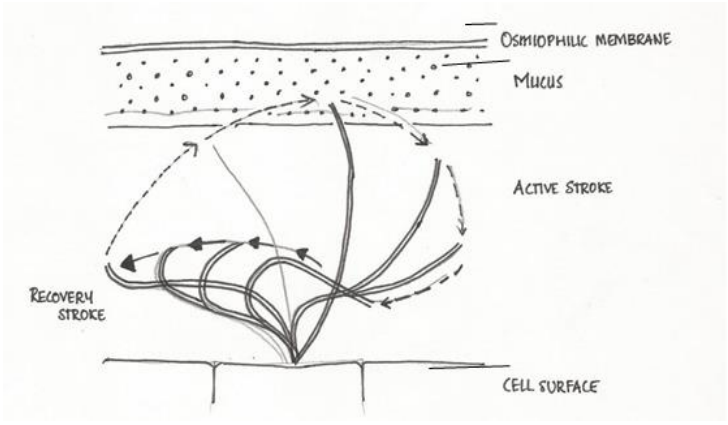


Figure 5: Normal ciliary cycle.

PTERYGOPALATINE FOSSA

The pterygopalatine fossa is a small pyramidal space below the apex of the orbit on the lateral aspect of the skull.[15]

The posterior boundary is the root of the pterygoid process and adjoining anterior surface of the greater wing of the sphenoid, the anterior boundary is formed by the superomedial part of the infra-temporal part of the maxilla. the lateral boundary is formed by the perpendicular plate of the palatine bone, with the orbital and sphenoidal processes forms the medial boundary, and the pterygomaxillary fissure is. Through the sphenopalatine foramen the fossa communicates with the nasal cavity, with the orbit via the medial end of the inferior orbital fissure, and with the infratemporal fossa via the pterygomaxillary fissure. The pterygomaxillary fissure which lies posterior to the maxilla and anterior to the pterygoid process of the sphenoid transmits the maxillary artery. Through the greater palatine canal, which opens in the posterolateral aspect of the hard palate pterygopalatine fossa also communicates with the oral cavity. There openings in the posterior wall of the PPF are two in number, the foramen rotundum, which transmits the maxillary nerve, and pterygoid canal, through which nerve of the pterygoid canal (Vidian nerve) runs. When the anterior aspect of pterygoid plate is studied in a disarticulated sphenoid, the foramen rotundum lies superior and lateral to the pterygoid canal.[15]

The main contents of PPF are the third part of maxillary artery and its 6 branches, the maxillary nerve and its branches, and the pterygopalatine ganglion.

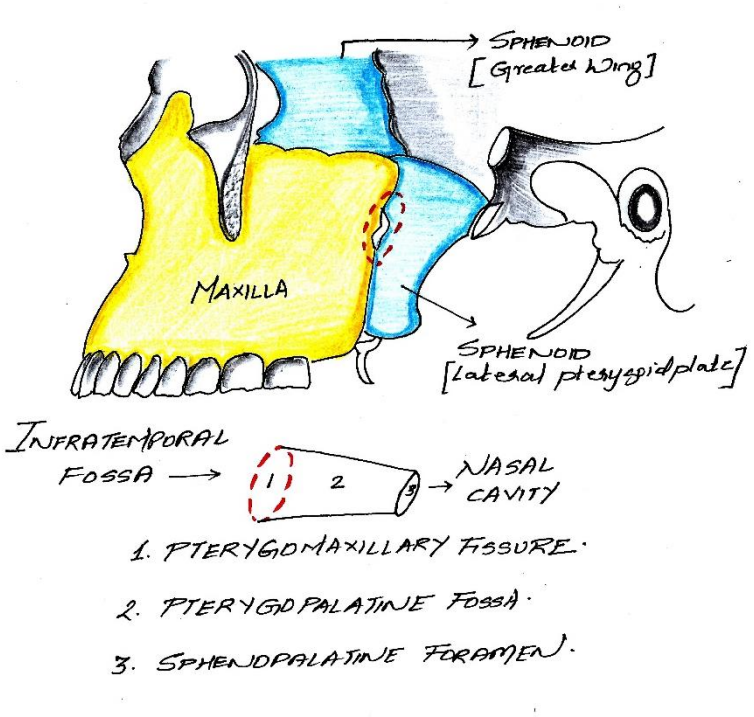


Figure 6: Relations of Pterygopalatine Fossa

MAXILLARY ARTERY

The maxillary artery courses through the pterygomaxillary fissure from the infratemporal fossa into PPF, where it ends as third part of the maxillary artery. It gives off six branches namely the posterior superior alveolar, infraorbital, sphenopalatine greater palatine arteries and the artery to pterygoid canal.[15]

Posterior Superior Alveolar Artery

The maxillary artery gives the posterior superior alveolar artery from within the PPF and runs through pterygomaxillary fissure onto the maxillary tuberosity. It gives off branches which penetrate the bone here to supply the maxillary, molar and premolar teeth and the maxillary antrum, and other branches that supply the buccal mucosa.

Occasionally the posterior superior alveolar artery arises from the infraorbital artery.[15]

Infraorbital Artery

The infraorbital artery enters the orbit via the inferior orbital fissure. It runs on the floor of the orbit in the infraorbital groove and infraorbital canal and emerges onto the face at the infraorbital foramen to supply the lower eyelid, side of the external nose, part of the cheek and the upper lip. While within the infraorbital canal it gives off the anterior superior alveolar artery which runs downwards to supply the anterior teeth and the anterior portion of the maxillary sinus. A middle superior alveolar artery can be present. When present, it branches from the infraorbital artery within the infraorbital canal and runs inferiorly along lateral wall of the maxillary sinus toward the region of canine and lateral incisor teeth and anastomoses with anterior and posterior superior alveolar arteries.[15]

Artery of The Pterygoid Canal

The artery of the pterygoid canal (Vidian artery) arises as a branch of either the third part of the maxillary artery (70%) or the petrous segment of the internal carotid artery (30%). It courses through the pterygoid canal and anastomoses with the pharyngeal, ethmoidal and sphenopalatine arteries in PPF and with the ascending pharyngeal, accessory meningeal, ascending palatine and descending palatine (occasionally) arteries in the oropharynx and around the pharyngotympanic tube. Through these complex anastomoses, the artery of the pterygoid canal contributes to the blood supply to segment of the pharyngotympanic tube, the tympanic cavity, and the upper part of the pharynx. It may also anastomose with the artery of the foramen rotundum, and so communicate with branches of the cavernous portion of the internal carotid artery.[15]

Pharyngeal Artery

Through the palatovaginal canal the pharyngeal division of the maxillary artery passes along with the nerve of the same name, and supplies the mucosa of the nasal roof, nasopharynx, sphenoidal air sinus and pharyngotympanic tube.

Greater (Descending) Palatine Artery

The greater palatine artery leaves the PPF through the greater (anterior) palatine canal, within which it gives off two or three lesser palatine arteries. The greater palatine artery supplies the inferior meatus of nose, then passes onto the roof of hard palate at the greater (anterior) palatine foramen and runs forwards to supply the hard palate and the palatal gingivae of the maxillary teeth. It gives a branch which runs up into the incisive canal to anastomose with the sphenopalatine artery and contributes to the arterial supply of the nasal septum. The lesser palatine arteries emerge onto the palate through the lesser (posterior) palatine foramen, or foramina, and supply the soft palate.[15]

Sphenopalatine Artery

The sphenopalatine branch of maxillary artery passes through the sphenopalatine foramen and enters the nasal cavity posterior to the superior meatus. From here its posterior lateral nasal branches ramify over the conchae and meatuses, anastomosing with the ethmoidal arteries and nasal branches of greater palatine artery to supply the frontal, maxillary, ethmoidal and sphenoidal air sinuses. The sphenopalatine artery next crosses anteriorly on the inferior surface of the sphenoid and terminates on the nasal septum in a series of posterior septal branches which anastomose with the ethmoidal arteries. The sphenopalatine artery also gives a small branch which runs along the inferior border of the inferior turbinate called the artery of Zuckerkandl. A branch descends on the vomer to the incisive canal to anastomose

with the greater palatine artery and septal branch of superior labial artery. [15]

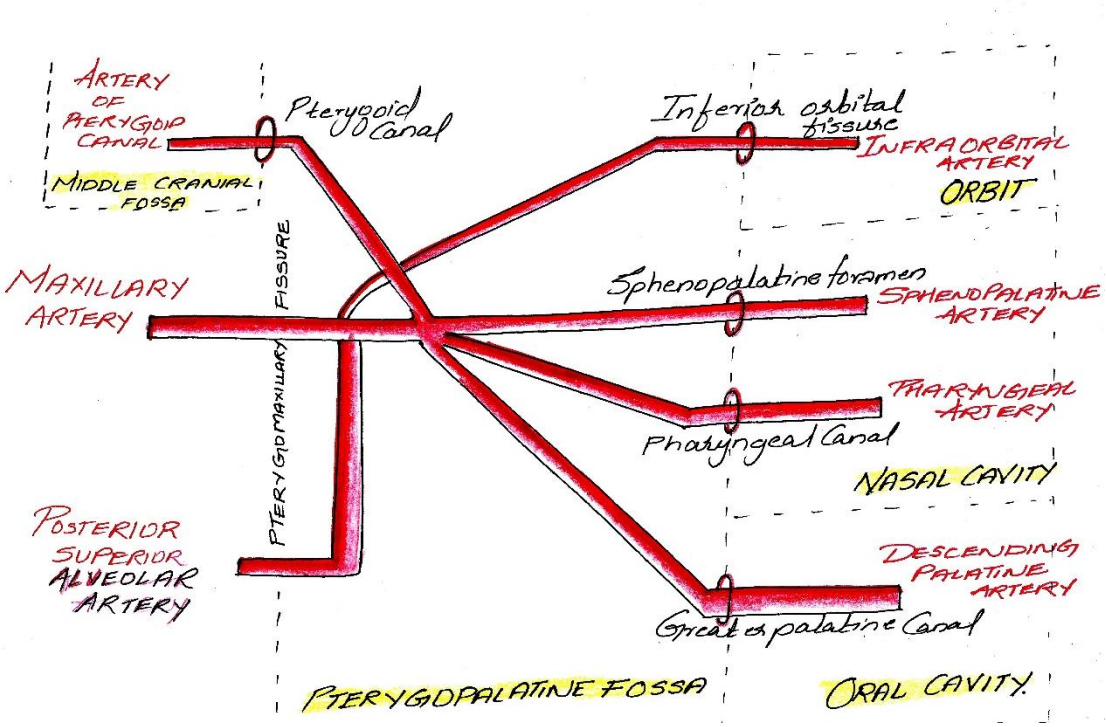


Figure 7: Schematic diagram of Maxillary artery in pterygopalatine fossa

MAXILLARY NERVE

The maxillary division of the trigeminal nerve is mainly sensory. It leaves the skull via the foramen rotundum, which leads directly into the posterior wall of the PPF. Crossing the upper part of the PPF, the nerve then gives two large ganglionic branches which contain fibres for the nose, palate and pharynx, and they pass through the pterygopalatine ganglion without synapsing. It then bends sharply laterally on the posterior surface of the orbital process of the palatine bone and on the upper part of the posterior surface of the maxilla in the inferior orbital fissure (which is continuous posteriorly with the PPF). It lies outside the orbital periosteum, and gives off its zygomatic, and then posterior superior alveolar branches. About halfway between the orbital apex and the orbital rim the maxillary nerve turns medially to enter the infraorbital canal as the infraorbital nerve.

The maxillary nerve gives many of its branches in the PPF. They can be subdivided into those that come directly from the nerve, and those that are associated with the pterygopalatine parasympathetic ganglion. Named branches from the main trunk are meningeal, ganglionic, zygomatic, posterior, middle and anterior superior alveolar and infraorbital nerves. Named branches from the pterygopalatine ganglion are orbital, nasopalatine, posterior superior nasal, greater (anterior) palatine, lesser (posterior) palatine and pharyngeal.[15]

Meningeal Nerve

The meningeal branch of the maxillary nerve arises within the middle cranial fossa and runs with the middle meningeal vessels. It innervates the dura mater.

Ganglionic Branches

There are usually two ganglionic branches that connect the maxillary nerve to the pterygopalatine ganglion.

Zygomatic Nerve

The zygomatic branch of the maxillary nerve leaves the PPF through the inferior orbital fissure together with the maxillary nerve.

Posterior Superior Alveolar Nerve

The posterior superior alveolar nerve leaves the maxillary nerve in the PPF.

Infraorbital Nerve

The infraorbital nerve is the terminal branch of the maxillary nerve. It leaves the PPF to enter the orbit at the inferior orbital fissure.

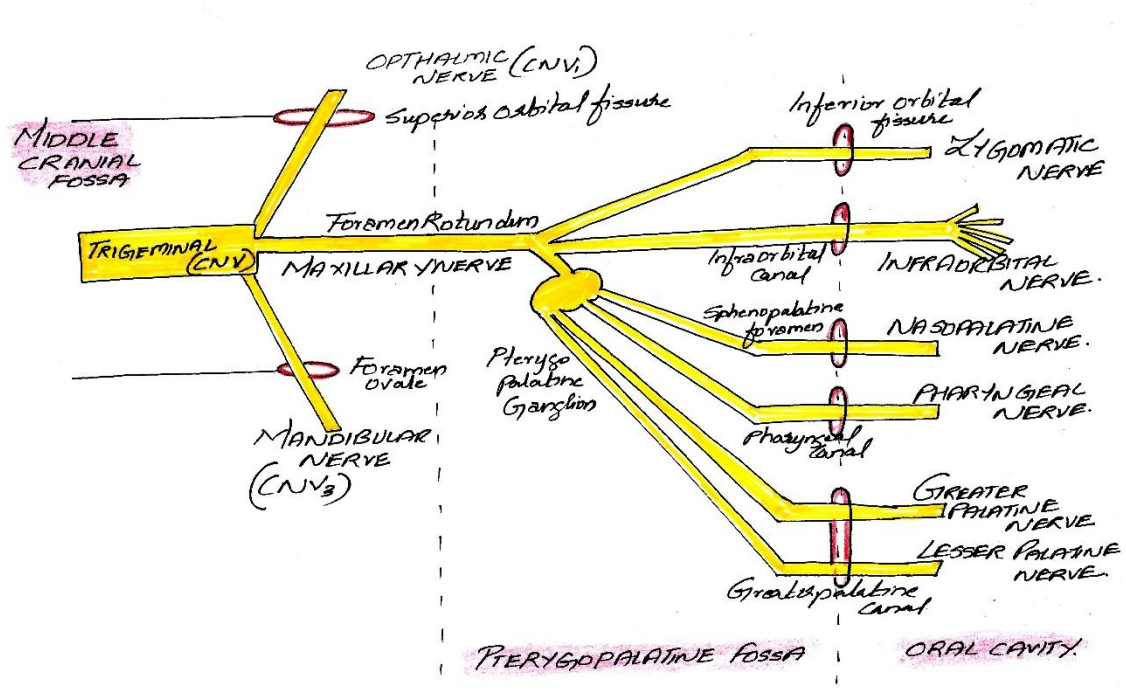


Figure 8: Schematic diagram of Maxillary Nerve in Pterygopalatine fossa

PTERYGOPALATINE GANGLION

The pterygopalatine ganglion is the largest of the peripheral parasympathetic ganglia. It is placed deep in the PPF, near the sphenopalatine foramen, and anterior to the pterygoid canal and foramen rotundum (Fig. 6). It is flattened, reddish grey in colour, and lies just below the maxillary nerve as it crosses the PPF. The majority of the 'branches' of the ganglion are connected with it morphologically, but not functionally, because they are primarily sensory branches of the maxillary nerve. Thus they pass through the ganglion without synapsing, and enter the maxillary nerve through its ganglionic branches, but they convey some parasympathetic fibres to the palatine, pharyngeal and nasal mucous glands.[15]

Preganglionic parasympathetic fibres destined for the pterygopalatine ganglion run initially in the greater petrosal branch of the facial nerve, and then in the nerve of the pterygoid canal (Vidian nerve), after the greater petrosal unites with the deep petrosal nerve. The nerve of the pterygoid canal enters the ganglion posteriorly. Postganglionic parasympathetic fibres leave the ganglion and join the maxillary nerve via a ganglionic branch, then travel via the zygomatic and zygomaticotemporal branches of the maxillary nerve to the lacrimal gland. Preganglionic secretomotor fibres of also travel in the nerve of the pterygoid canal. They synapse in the pterygopalatine ganglion, and postganglionic fibres are distributed to palatine, pharyngeal and nasal mucous glands via palatine and nasal branches of the maxillary nerve.[15]

Postganglionic sympathetic fibres pass through the ganglion without synapsing and supply blood vessels and orbit. They arise in the superior cervical ganglion and travel via the internal carotid plexus and deep petrosal nerve to enter the pterygopalatine ganglion within the nerve of the pterygoid canal.[15]

General sensory fibres destined for distribution via orbital, nasopalatine, superior alveolar, palatine and pharyngeal branches of the maxillary division of the trigeminal nerve run through the ganglion without synapsing.

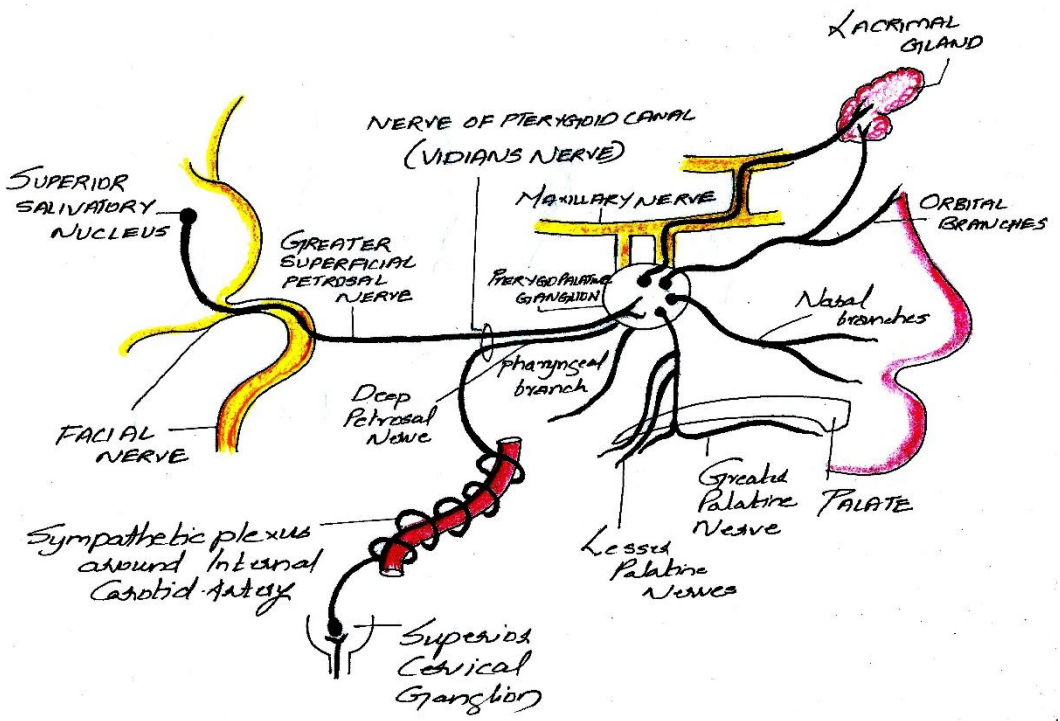


Figure 9: Pterygopalatine Ganglion

Orbital Branches

Fine orbital branches enter the orbit through the inferior orbital fissure and supply orbital periosteum. Some fibres also pass through the posterior ethmoidal foramen to supply the sphenoidal and ethmoidal sinuses. The orbital branches join branches of the internal carotid nerve to form a 'retro-orbital' plexus from which orbital structures such as the lacrimal gland receive an autonomic innervation.

Nasopalatine Nerve

The nasopalatine nerve leaves the PPF through the sphenopalatine foramen and enters the nasal cavity. It passes across the cavity to the posterior part of the nasal septum, runs downwards and forwards on the septum in a groove in the vomer, and

then turns down through the incisive fossa in the anterior part of the hard palate to enter the roof of the mouth. When an anterior and a posterior incisive foramen exist in this fossa, the left nasopalatine nerve passes through the anterior foramen, and the right nerve passes through the posterior foramen. The nasopalatine nerve supplies the lower part of the nasal septum and the anterior part of the hard palate, where it communicates with the greater palatine nerve.[15]

Posterior Superior Nasal Nerves (Lateral and Medial)

The posterior superior alveolar nerves enter the nasal cavity through the sphenopalatine foramen. Lateral posterior superior nasal nerves (about 6) innervate the mucosa lining the posterior part of the superior and middle nasal conchae and the posterior ethmoidal sinuses. Two or three medial posterior superior nasal nerves cross the nasal roof below the opening of the sphenoidal sinus to supply the mucosa of the posterior part of the roof and of the nasal septum.

Palatine Nerves (Greater and Lesser)

The greater and lesser palatine nerves pass downwards from the pterygopalatine ganglion through the greater palatine canal. The greater palatine nerve descends through the greater palatine canal, emerges on the hard palate from the greater palatine foramen and runs forwards in a groove on the inferior surface of the bony palate almost to the incisor teeth. It supplies the gingivae, mucosa and glands of the hard palate and also communicates with the terminal filaments of the nasopalatine nerve. In the greater palatine canal, it gives off posterior inferior nasal branches that emerge through the perpendicular plate of the palatine bone and ramify over the inferior nasal concha and walls of the middle and inferior meatuses. As it leaves the greater palatine canal, it gives off branches which are distributed to both surfaces of the adjacent part of the soft palate.

The (middle and posterior) palatine nerves are much smaller than the greater palatine nerve. They descend through the greater palatine canal from which they diverge low down to emerge through the lesser palatine foramina in the tubercle or pyramidal process of the palatine bone. They innervate the uvula, tonsil and soft palate.[15]

CHRONIC RHINOSINUSITIS

The term "sinusitis" refers to a group of disorders characterized by inflammation of the mucosa of the paranasal sinuses. Because the inflammation nearly always also involves the nasal mucosa, it is now called "rhinosinusitis".[17]

To highlight the role of inflammation better, newer definitions have been applied to rhinosinusitis that is, a group of disorders characterized by inflammation of the mucosa of the nose and paranasal sinuses. Chronic rhinosinusitis (CRS) is rhinosinusitis of at least 12 consecutive weeks duration.[18]

Therefore, CRS is a group of disorders characterized by inflammation of the mucosa of the nose and paranasal sinuses of at least 12 consecutive weeks duration.[18,19]

A widely accepted set of classifications or definitions was developed by the Rhinosinusitis Task Force of the American Academy of Otolaryngology-Head and Neck Surgery and reported by Lanza and Kennedy.[18,20,21]

Table 1. Classification of Rhinosinusitis

CLASSIFICATION	DURATION
Acute (ARS)	7 Days to <4 weeks
Sub-acute	4-12 weeks
Recurrent acute	> 4 episodes of ARS per year
Chronic (CRS)	>12 Weeks
Acute exacerbation of chronic	Sudden worsening of CRS with return to baseline

The taskforce in order to accommodate the different needs gave definitions that can be applied in appropriate studies. In this way the taskforce tried to improve the

comparability of studies and gave evidence-based diagnosis and treatment of patients with rhinosinusitis and nasal polyps.

CRS is best defined as a group of heterogeneous disorders due to a multitude of causes that result in mild to severe symptomatic inflammation of the sinonasal mucosa. The management of this complex disease is therefore a challenge. The most simplified classification divides CRS into those patients who have nasal polyp (CRSwNP) and those without (CRSsNP) .[17]

Table 2. CRS differentiation by inflammatory mediators.[17]

CRS with nasal polyps (CRSwNP)	Tissue oedema, low tumour growth factor -β and lot T-reg activity High tissue eosinophillia and IgE increased IL-5 and IL-13 (Th2 polarisation)
CRS without nasal polyps (CRSsNP)	Fibrosis, less eosinophillic infiltration Increased interferon -ɣ, tumour growth factor-β and T--regulatory activity (Th1 polarisation)

Although all cases of rhinosinusitis involve inflammation of the mucosal linings, the focus is on those patients in whom this inflammation leads to symptoms. Because of this important relationship to symptoms, the Rhinosinusitis Task Force's definitions include a group of symptoms to be applied to these conditions to allow for clinical diagnosis.

Rhinosinusitis symptoms/signs (requires two major factors, or one major and two minor).

Table 3. Symptoms of Chronic rhinosinusitis.[5]

MAJOR SYMPTOMS	MINOR SYMPTOMS
Facial pain /Pressure	Headache
Facial congestion/Fullness	Fever (Non acute)
Nasal Obstruction /Blockage	Halitosis
Nasal discharge/Purulence/Discoloured Posterior drainage	Fatigue
Hyposmia/Anosmia	Dental pain
Purulence on nasal examination	Cough
Fever (Acute RS only)	Ear pain/Pressure/Fullness

Epidemiology

CRS is the fifth most common diagnosis for an antibiotic prescription worldwide.[5,16]. Despite its prevalence, there is a paucity of accurate epidemiologic data for CRS, especially for CRSsNP. Patient surveys in the United States have found a 15%–16% prevalence of CRS. A study conducted in Canada, Korea, Scotland, Europe, and Brazil show that prevalence of CRS ranges from 1%–11%. [5,17,18]

Men and women are both affected by CRSwNP. In general, nasal polyps occur in all races and become more common with age, with the average age of onset being 42 years.

Etiology

Numerous hypotheses have been proposed with a great deal of overlap, supporting a multifactorial etiology. One classification method separates potential contributing entities into host and environmental factors. The heterogeneous nature of CRS is important to understand when planning treatment for this diverse group of patients

whose disease may have arisen from very different underlying etiologies.[5]

Table 4. Factors associated with CRS.[5]

SYSTEMIC HOST FACTORS	LOCAL HOST FACTORS	ENVIORMENTAL FACTORS
1) Allergy	1) Anatomic	1) Microorganism (bacteria, fungi, virus)
2) Immunodeficiency	2) Neoplasm	2) Noxious chemicals
3) Mucociliary dysfunction	3) Acquired mucociliary dysfunction	3) Medications
4) Cystic fibrosis	4) Previous trauma or Surgery	
5) Granulomatous diseases		
6) GERD		
7) Aspirin intolerance		

CRSwNP in the Caucasian population is associated more closely with high tissue eosinophilia and increased T helper (Th)-2 cytokine expression (interleukin [IL]-5 and IL-13) as well as nasal obstruction and hyposmia, whereas CRSsNP may have more Th-1 polarization and less eosinophilic infiltration.

Diagnosis

At the first consultation, the diagnosis of rhinosinusitis is presumed on symptoms alone. The symptoms are mainly the same in acute rhinosinusitis (ARS), CRSsNP and CRSwNP, but the pattern and intensity may vary. Litvack et al in their study reported a significantly increased risk of hyposmia (odds ratio = 2.4 and anosmia (Odds ration =13.2) in nasal polyposis patients compared to CRSsNP. (14) After inquiring the symptoms, anterior rhinoscopy remains the first step in clinical examination, though it is of limited value .[6]

Examination

i. ***Nasal endoscopy***

Nasal endoscopy involves passing a rigid or sometimes, flexible, endoscope through the nostril to examine the nasal cavity, middle and superior meati, nasopharynx and mucociliary drainage pathways. Nasal endoscopy has a major contribution in the diagnosis of CRS and affords much better illumination, magnification and visualization of the nasal cavity compared to anterior rhinoscopy.[6]

ii. ***Imaging***

The plain sinus x-ray has limited usefulness for the diagnosis of rhinosinusitis and for evaluation of the response to therapy. CT scanning is the modality of choice for the paranasal sinuses due to optimal display of differences between air, bone and soft tissue. CT scanning is not the primary step in the diagnosis of rhinosinusitis but has the aim to confirmand correlate the symptoms and findings of endoscopic examination after failure of medical therapy. Because of many insignificant abnormalities found in the normal population during scans.[15], the diagnosis of CRS based on imaging, in absence of symptoms, is inappropriate.[6]

iii. ***Nasal cytology, biopsy and bacteriology***

Cytology is not a useful tool in diagnosis of rhinosinusitis. However, lavage with 0.9% saline, micro suction nasal brushes, nasal tampons, disposable scrapers, etc. are techniques which can be used for diagnosis.[6]

CRS is diagnosed based on clinical symptoms and objective evaluation. Symptoms must be present for at least 12 consecutive weeks. Several studies have shown using symptoms alone to diagnose CRS can be nonspecific. Therefore, nasal endoscopy or imaging must also be used to confirm the presence of sinonasal disease to increase

the specificity of diagnosis.[6]

Endoscopic findings suggestive of CRS include mucopurulent discharge, nasal polyps or polypoid change, and/or mucosal edema obstructing the middle meatus

Computed tomography (CT) is the gold standard for imaging in CRS. Although CT scans cannot distinguish between inflammation and infection, they show fairly well the extent of disease. Findings consistent with CRS include isolated or diffuse mucosal thickening, bone changes, or air-fluid levels.[15]

Treatment

Aim of the treatment is to improve osteomeatal complex drainage.

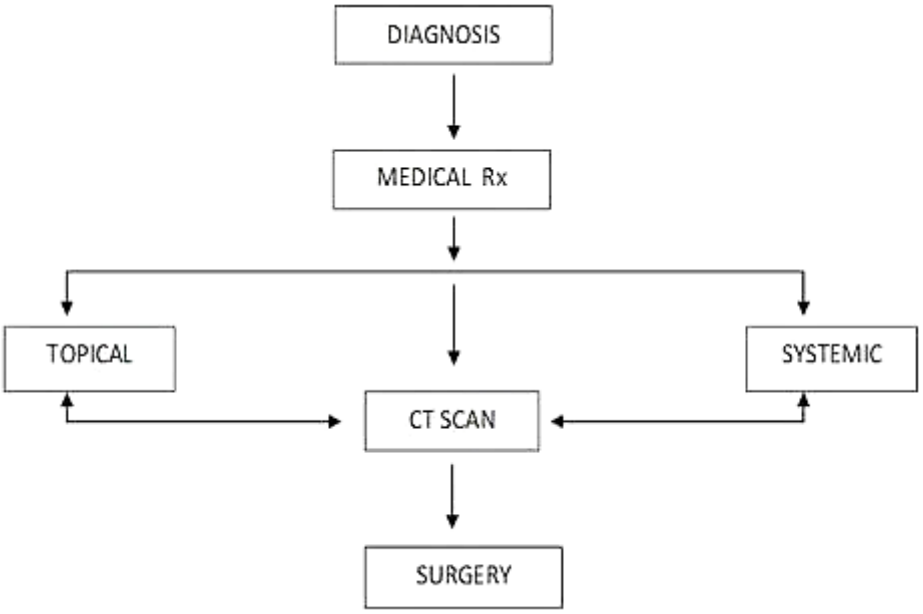


Figure 10: Treatment Algorithm

Medical treatment. [5]

Medical treatment involved in CRS includes:

1) Allergen and/or irritant avoidance
2) Douching
3) Corticosteroids
4) Decongestants
5) Antibiotics
6) Antifungals
7) Antileukotrienes
8) NSAID
9) Immunotherapy
10) Other therapies

Corticosteroids

Systemic corticosteroids (oral, intramuscular) can reduce the size of nasal polypi to some extent that is compatible with surgery. Topical corticosteroids reduce the recurrence of nasal polypi and should be used routinely in the long term, preferably employing a molecule with low systemic absorption in drop form in the head down position in non-polypoid chronic rhinosinusitis, topical corticosteroid reduces symptoms during acute exacerbations when combined with antibiotics.

Antibiotics

Antibiotics are needed for acute severe bacterial sinusitis; their place in the chronic form is controversial.

Two routes are available: topical and oral.

Oral short course is used in the treatment of acute exacerbations of CRS.

Antihistamines /Antileukotrienes

There is limited benefit in using antihistamines in chronic rhinosinusitis, because the majority have little or no effect on nasal blockage.

Leukotriene antagonists, such as Montelukast, Zafirlukast, and Zileuton, have been evaluated in numerous studies involving patients with CRSwNP

Surgical treatment

Functional endoscopic sinus surgery (FESS)

FESS has now become well established for the treatment of chronic rhinosinusitis refractory to medical treatment

Steps:

I. Uncinectomy
II. Middle meatal antrostomy
III. Anterior Ethmoidectomy
IV. Posterior ethmoidectomy
V. Clearance of frontal recess and frontal sinusotomy
VI. Sphenoidotomy

Table 5. Complications of FESS[21]

SITE	COMPLICATION
Orbit	Nasolacrimal duct damage Extra-occular muscle injury Intra-orbital haemorrhage/ Emphysema Optic nerve damage
Intracranial	Haemorrhage Cerebrospinal fluid leak--> meningitis
Nasal	Haemorrhage

NASAL POLYPOSIS

Nasal polyps (NP) are benign lesions arising from the mucosa of the paranasal sinuses or from the mucosa of the nasal cavity. Having an uncertain etiology and a tendency to recur, they are a challenging diagnosis to treat.

Etiology of nasal polyposis

The etiology of NP is unknown. Some theories suggest it as a consequence of conditions which cause chronic inflammation in the nose and paranasal sinuses characterized by stromal edema and variable cellular infiltrate the local allergic mechanisms in the absence of systemic features play a role in the pathogenesis of polyps.[22]

Interleukin -5 (IL-5) has found to be significantly raised in NP compared with healthy controls and the concentration of IL-5 was independent of the atopic status of the patient.[23] The key role of IL-5 was supported by the finding that treatment of eosinophil-infiltrated polyp tissue with neutralizing anti-IL 5 monoclonal antibody resulted in eosinophil apoptosis.[24] The regulation of the IL-5 receptor has also been investigated with down regulation being found to occur in NP, especially in association with asthma.[25]

A link has been demonstrated recently between HLA-A74 and NP, but the current knowledge in this area remains very limited.[26]

Medical conditions commonly associated with polypi include asthma, bronchiectasis, and cystic fibrosis .[27]

Clinical features

Symptoms of NP most commonly are

a) nasal obstruction which is constant but can vary depending on the site and size of the polyps.
b) watery rhinorrhea and postnasal drip.
c) Anosmia or hyposmia with an ensuing alteration in taste[28]

Anterior and posterior rhinoscopy reveals single or multiple pale, grey polypoid masses arising most frequently from the middle meatus and prolapsing into the nasal cavity. They consist of loose connective tissue, edema, inflammatory cells, and some capillaries and glands. They are covered with different types of epithelium, most commonly pseudostratified respiratory epithelium with goblet cells and ciliated cells. Studies have shown that eosinophils are the most common inflammatory cells in NP. This leads to a large proportion of IL-5 due to prolonged eosinophil survival and this is one of the important features in differentiating NP from rhinosinusitis at a histo-chemical level.[29]

NP are almost invariably bilateral and when unilateral require histological examination to exclude malignancy or other pathology such as inverted papilloma. They are insensitive to palpation and rarely bleed.[30]

The histological appearance of NP is characterized by ciliated columnar epithelium, thickening of the basement membrane, a loose avascular edematous stroma and an infiltrate of plasma cells and eosinophils. Eosinophils are found in 85% of NP with the remaining cells being predominantly neutrophils.[31]

Investigations

A CT scan will show the extent of NP and anatomical variations and is essential if surgical treatment is being planned. It is not the primary step in the diagnosis of the condition, except where there are unilateral signs and symptoms or other sinister features. It corroborates history and endoscopic findings after failure of medical therapy.

A range of staging systems for CT scanning have been described, the most commonly used being the Lund-Mackay system.[32] This system relies on a score of 0–2 dependent on the absence, partial, or complete opacification of each sinus system and of the vital osteomeatal complex deriving a maximum score of 12 per side. This has been validated but the correlation between the CT score and symptoms has been shown to be poor and is not a good indicator of outcome .[33]

In unilateral cases of NP, a magnetic resonance imaging scan may help in diagnosis, particularly for investigations of more serious conditions such as neoplasia.

Treatment of NP

Therapy for NP involves a combination of observation, medical, and surgical treatments depending on individual case assessment

The aims of treatment are to eliminate or significantly reduce the size of the NP resulting in relief of nasal obstruction, improvement in sinus drainage, restoration of olfaction and taste.

Medical treatment

1) Corticosteroids

Corticosteroids form the mainstay of conservative therapy in NP as both a primary treatment and to prevent recurrence Their clinical efficacy is achieved by a combination of anti-inflammatory effects along with their ability to reduce airway eosinophilic infiltration by preventing their increased viability and activation. Both topical and systemic glucocorticoids affect the eosinophil function by both directly reducing eosinophil viability and function or indirectly reducing the secretion of chemotactic cytokines by nasal mucosa and polyp epithelial cell.[34] In the absence of other warning signs such as pain, bleeding, or unilateral polyps, treatment can be done in primary care setting. Corticosteroids should be used with caution in 'at-risk groups' particularly patients with diabetes, uncontrolled hypertension, and peptic ulcer disease.

Initially topical nasal steroids are delivered by drops or sprays along with aggressive treatment of any underlying cause or comorbid allergy. Inspite of symptomatic benefit, there is controversy in the efficacy of steroids in reduction of the proportion of patients requiring surgery.

Systemic steroids are reserved for advanced or refractory cases particularly when allergy is present. This form of treatment is used for relatively rapid short-term improvement due to the risk of adverse effects

2) Saline Nasal Douching

These irrigations have been shown to improve nasal mucocilliary clearance measured by the saccharine test in both NP and healthy volunteers in an American study.[35]

3) Mucolytics can be used as adjuncts to antibiotics in acute sinusitis to reduce viscosity of sinus secretion but no clinical trials have tested their effects in NP.
4) Antihistamines can have significant reduction in symptoms in simple NP.
5) Other medical therapies

a) Leukotriene receptor antagonists have been shown to be effective [36]
b) Topical capsaicin has also been shown to be effective, but side-effects including burning of the nasal mucosa limit its acceptability to patients[37]

Surgical treatment

Surgical therapy is reserved for cases refractory to medical treatment.

ESS involves restoring sinus drainage by careful removal of NP or other soft tissue obstructing the natural sinus ostia. This can be done by traditional snare polypectomy, cold steel instruments, or the increasingly more common technique of micro-debridement, which involves a rotating blade in combination with suction and irrigation.[38]

Postoperatively as a routine part of after-surgery care to prevent

a) crusting and adhesions-regular saline nasal douching
b) recurrence-Topical intranasal steroids

ALLERGIC RHINITIS

Allergic rhinitis is an Ig E mediated hypersensitivity disease of the mucous layer of the nasal cavity characterized by sneezing, itching, watery nasal discharge and sensation of nasal obstruction.[39] The diagnosis of inhalant allergy can be made when history, physical examination and skin test/RAST results are combined.[39,40]

EPIDEMIOLOGY AND ETIOLOGY OF ALLERGIC RHINITIS

The development of allergic rhinitis depends on

i. Atopic state of sensitivity to an allergen.

ii. Exposure of sensitized subject to the allergen.[41]

iii. The principal cause of allergic rhinitis is sensitivity to inhalant allergens.[40]

PATHOGENESIS OF ALLERGIC RHINITIS

Allergic rhinitis is an immediate hypersensitive Ig E mediated response of the nasal mucosa activating inflammatory process. When an allergen intrudes the body, it is bound by antigen presenting cells and is presented as antigen - Major histocompatibility Complex-II complex to T lymphocytes.[41,42,43,44]

TREATMENT

Non-pharmacological treatment [45,46]

1) Prophylactic measures like avoidance of the allergen
2) Frequent washing of the hands
3) Keeping the hands away from the eyes, nasal mucosa
4) Keep fur of pets away from the home
5) Avoidance of mechanical rubbing of the nose to reduce mast cell degranulation

6) Cold compress to reduce allergic inflammation and rhinitis discomfort through vasoconstriction.

7) Geographical relocation

Pharmacological treatment:

Anti-allergens can be classified as:

I. Inhibiting synthesis or release of prostaglandins, histamines and leukotrienes.[47,48,42,49,50,43]

II. Agents inhibiting chemical mediators from their target sites. [47,41,42,43]

Pharmacological agents are[47,41]

a) Mast cell stabilizers

b) Antihistamines

c) Anti-inflammatory drugs

d) Immunosuppressive agents

e) Leukotriene antagonists

f) Subcutaneous Immunotherapy: It involves repeated administration of subcutaneous injections of a particular antigen one person is sensitized for. In the beginning it is started with minute doses and is increased gradually to larger doses until desensitization is achieved. This increases the Ig G production or T cell mediated suppression of allergic response. The effectiveness of this modality in seasonal allergic rhinitis is mentioned in literature.[51]

g) Sublingual immunotherapy for allergic rhinitis: Sublingual immunotherapy for allergic are under trials now.[52]

Surgical Management

B/L inferior turbinoplasty

INFERIOR TURBINATE HYPERTROPHY

Hypertrophy of the inferior turbinate occurs when there is dilatation of the venous sinusoids, deviated nasal septum on the opposite side as compensatory hypertrophy, hypertrophy of the lamina propria (medial end), oedema due to local inflammation or due to enlargement of bone itself. Sympathetic and parasympathetic activity controls this in whole as the turbinates have rich blood supply.[53]

Surgical Treatment:

Management of rhinitis where medical modality has failed is by surgery. Good ventilation and drainage are needed for the normal functioning of mucosal layer of nasal cavity and sinus mucosa which can be affected by structural deformities that reduce normal function or contributes to significant nasal obstruction. Therefore, these deformities should be addressed. Commonest procedure performed to reduce nasal obstruction is on inferior turbinate reduction.[54] Numerous surgical procedures have been designed to make the inferior turbinate small or remove a portion or even the entire inferior turbinate. Surgical options address the bone, submucosa, mucosa or a combination of these. Most procedures can be performed under local anesthesia.[54,55] The most common surface mucosal procedure is cryotherapy, in which a cryoprobe is placed along the medial surface for time period of seconds to a minute or more. The temperature of probe tip is approximately -70 degree.

A very low temperature causes formation of fibrotic tissue due to vessel thrombosis, which prevents the venous sinusoids in the turbinate mucosa from swelling up as a response to various allergens.[56,57,58] Submucosal cauterization is performed by

placing a needle into the submucosal tissues of the inferior turbinate and applying an electrical current for various periods of time until blanching of the mucosa is visualised. Mechanism of action in submucosal cauterization is similar to that of cryothearpy. It is an easy and well tolerated procedure to reduce turbinate size and has said to improve nasal obstruction symptom but for minimal period of time. Both cryotherapy and submucosal cauterization may need repeated attempts for symptom free period. Both these techniques are known to cause significant nasal crusting for a week or more post operatively. However saline nasal douching can bring down the severity of this problem.[59]

Radiofrequency ablation is a newer technique which is similar to that of cauterization. The radiofrequency probe is inserted submucosally into the inferior turbinate and turned on for various periods of time. The radiofrequency generated heat ablates tissue and effectively shrinks the inferior turbinate. In a study by LIN Hsin-Ching used radiofrequency in the treatment of allergic rhinitis for the patients who were unresponsive to medical therapy. The visual analogue scores for allergic rhinitis symptoms showed significant reduction up to 1year after surgery. [59,60]

Inferior turbinoplasty is a procedure usually performed under general anaesthesia and is designed to remove bone and some of the submucosal tissue. Turbinate resection involves partial or rarely full removal of the inferior turbinate. However, complete resection increases chance for atrophic rhinitis, so the procedure is obsolete now. Anterior end of the inferior turbinate is preserved to retain the normal functions. Resection includes bony turbinate and mucosa, humidification, air filtration and warming of the inhaled air can be preserved which are the normal physiological functions of nasal mucosa.[61]

Among patients suffering from chronic nasal obstruction, postoperative symptoms

improved with regards to the clinical features of nasal block, sneezing, nasal discharge, and mouth breathing at 1, 2, and 3 years after surgery with radiofrequency ablation of the inferior turbinates.

Rhinomanometric assessment also showed significant improvement at 1, 2, and 3 years postoperatively ($P < .05$ for all). Saccharin transit time was significantly decreased ($P < 0.05$ for all) compared to preoperative values at 1, 2, and 3 years after surgery.[60] Nasal breathing improved post submucosal diathermy by 89%.This has showed 78% success rate at one year postoperatively. Similar studies showed 87% improvement in nasal obstruction at 1 year & 77% at 2-5 years by comparing partial inferior turbinectomy and cryosurgery in nasal obstruction for cryosurgery group.[62] At 10 years analysis in a study 82% success rate was seen after inferior turbinectomy in patients of allergic rhinitis.[63]

However due to the rich blood supply of the turbinates, bleeding is one of the most common complications during turbinoplasty.

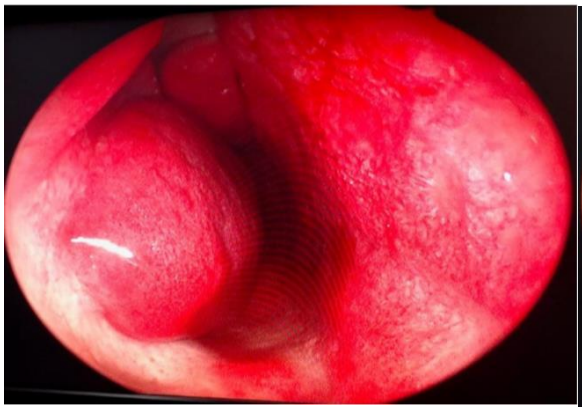


Figure 11: Inferior turbinate hypertrophy

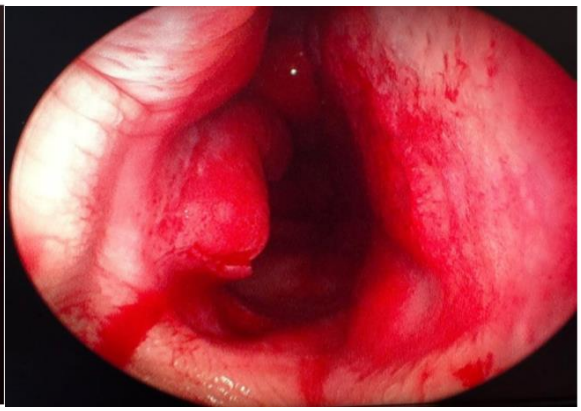


Figure 12: Post inferior turbinoplasty

CONTROL OF BLEEDING DURING ENDOSCOPIC NASAL AND PARANASAL SINUS SURGERIES

The presence of significant bleeding in the surgical field is a critical factor in the potential success or failure of endoscopic nasal surgery. When significant bleeding is present, recognition of anatomical landmarks becomes difficult. [64,65,66]

Bleeding obscures surgical planes and makes the identification of the drainage pathways of the sinuses difficult. Cell walls become difficult to distinguish from the lamina papyracea or skull base and the risk of causing complications increases.[65,66]

If the patient has significant inflammation of the sinuses, from chronic infection or the presence of pus/fungal debris, increased vascularity will often contribute to more bleeding.[64,67] If the surgeon attempts to manipulate an instrument in the surgical field after the anatomic landmarks are covered in blood, the risk of a complication increases. In addition, greater surgical trauma may occur, cells may be left behind and there is an increased likelihood of postoperative scarring and failure of the surgical procedure. It is therefore critical to optimize the surgical field and, in so doing, make the surgical dissection as easy as possible.[64,65,66]

Bleeding is more common close to large vessels. Stamberger has included 3 areas which are responsible for extensive bleeding during sinus surgery.

1. Anterior ethmoidal artery located in an osseous channel close to ethmoid roof Branch of sphenopalatine artery close to the posterior end of middle turbinate.
2. This is more prone for injury in patients with well pneumatized middle turbinate (concha bullosa)
3. Damage to sphenopalatine artery while attempting to widen the sphenoidal ostium

Bleeding during endoscopic nasal surgery has been classified into: Arterial, Venous and Capillary. Out of these three types of bleeding it is the capillary bleed that causes trouble most often during endoscopic nasal surgery.[68]

Different methods have been tried to control bleeding during sinus surgery, which are [69]

i. Controlled Hypotension

Controlled hypotension technique includes various modalities associated with different potency and adverse effects. Its safety is dependent on a thorough knowledge about the mechanism of action for each modality, adequate monitoring of the patient, and choosing the appropriate modality with consideration to history of drug allergies and co-morbidities.

ii. Patient's Positioning

Reverse Trendelenburg or anti-Trendelenburg position is a common surgical position in which the head is up, and feet are down. Head elevation reduces mean arterial pressure in the elevated part by about 2 mm Hg for each 2.5 cm above the cardiac level.[70] A reverse Trendelenburg position is used in numerous surgical procedures and presents multiple benefits. It reduces venous return from the lower extremities, therefore, reducing total blood loss, blood loss per minute ($P < 0.001$) and improving haemostasis of the surgical field when compared with the supine position.[71] Sudden shift in blood pressure is a serious complication of the reverse Trendelenburg position; the patient must be tilted in and out slowly to avoid this complication.[69]

iii. Using a Laryngeal Mask Airway

The laryngeal mask airway (LMA) is a supraglottic device that is associated with less respiratory and cardiovascular reflex responses due to reduced stimulation of the larynx as compared to endotracheal intubation. Moreover, LMA facilitates controlled hypotension. One study suggested that LMA is more effective than endotracheal intubation in regard to rapid onset to achieve a target systolic arterial blood pressure ($P< 0.05$), less blood loss), and use of lower doses of remifentanil ($P < 0.05$). Visibility of the operative field improved in the first 15 minutes ($P < 0.05$).[72]

iv. Technique of Ventilation

Ventilation with normocapnia or mild hypocapnia has been advocated to minimize bleeding and optimize the surgical field during endoscopic sinus surgery Mode of ventilation is important to control hypotension. Traditional intermittent positive pressure ventilation (IPPV) has a troublesome hemodynamic effect due to high intra-thoracic pressures and reduced venous return to the heart.[73] This result provides decreased blood circulation from the upper part of the body; therefore, it is a high risk for intra-operative bleeding. Conversely, high-frequency jet ventilation is small volume ventilation that provides adequate gas exchange at a lower pressure than intermittent positive pressure ventilation. A recent study compared efficacy between intermittent positive pressure ventilation and high frequency jet ventilation demonstrating that the total mean blood loss in the high-frequency jet ventilation group (170 ml) was significantly lower than the intermittent positive pressure ventilation group (318.8 ml; $P = 0.017$). The quality of the surgical field in high-frequency jet ventilation was significantly better than the intermittent positive pressure ventilation group ($P = 0.012$) .[74]

Medications for Controlled Hypotension[69]

i. Inhalation Anaesthetics

Controlled hypotension with inhalation agents (e.g., Isoflurane, Sevoflurane, and Desflurane) decreases arterial blood pressure through peripheral vasodilatation due to blockage of α-Adrenoceptors. However, higher concentrations can increase cerebral blood flow, increase the intracranial pressure, and deteriorate cerebral auto regulation. Therefore, a combination of inhalation Anaesthetics with other drugs is required to help reduce the concentration and adverse effects of each agent.[75]

ii. Intravenous Anaesthesia

Intravenous anaesthesia has been introduced for analgesia, hypnosis, sedation, and general anaesthesia (induction phase or maintenance phases). Agents currently used for general anaesthesia include propofol and opioids. Propofol has a depressant effect on the central nervous system via direct activation of the gamma-aminobutyric acid (GABA-A) receptors, inhibition of the n-methyl d-aspartate (NMDA) receptor, and modulation of the calcium influx through slow calcium ion channels. Propofol has rapid onset of action and recovery time with a dose-related effect. However, dose-dependent hypotension is its most common complication, especially high-dose infusions are associated with propofol infusion syndrome. This condition is a potentially fatal complication with severe metabolic acidosis and circulatory collapse. Traditional opioids have been used as analgesic drugs and they bring some hypotensive effect. However, this effect is difficult to use for controlled hypotension due to their long half-life. Remifentanil is a new potent ultra short-acting μ opioid agonist with a short half-life; therefore, its action has a rapid onset and offset. It

offers a reduction in sympathetic nervous system tone and dose-dependent effects, decreasing heart rate and blood pressure. In general, anaesthesia, opioids are often used as an adjunct of intravenous-based technique (opioids combined with antihypertensive drugs or propofol) or inhalation-based technique (opioids combined with an inhalation agent). There is controversy regarding efficacy of intravenous- or inhalation-based technique for controlled hypotension.[69]

iii. **Antihypertensive Drugs**

Antihypertensive drugs have numerous classifications and mechanisms of action to control blood pressure.[76] Controversy exists regarding which is the ideal mean arterial pressure (MAP) in controlled hypotension to reduce bleeding and the correlation between blood loss and MAP. MAP at a very low level does not correlate with decreased intra-operative blood loss. However, severe hypotension may further reduce blood supply to vital organs.[69]

Surgical Considerations

There are many methods to deal with intra-operative bleeding during endoscopic sinus surgery. Their choice often depends on whether the bleeding is from venous or arterial origin, the size of the vessel, and its location.[77]

1. Topical Vasoconstrictors.

The aim of topical vasoconstrictors is to decongest the nasal cavity, thus widening the nasal corridor and minimizing bleeding. Commonly used topical vasoconstrictors include cocaine, epinephrine, phenylephrine, and oxymetazoline. All topical vasoconstrictors have potential adverse effects; therefore, the property of each agent should be considered.[69]

2. Local Anesthetic with Vasoconstrictor Injection.

Infiltration of a solution of local anaesthetic with vasoconstrictor has been introduced to minimize intraoperative bleeding. Haemostatic efficacy of local anaesthetic with vasoconstrictor was demonstrated in a study that showed decreased bleeding when lidocaine/epinephrine was injected as compared to injection of placebo ($P < 0.05$).[78] However in another study the intraoperative estimated blood loss was not significantly different between the anaesthetic/epinephrine and control groups ($P > 0.05$) .In another study conducted by Wormald et all it was found that direct injection of, epinephrine into pterygopalatine fossa (containing the main nasal arterial supply) results in significantly better haemostasis ($P= 0.01$).[1] Arterial pressure and heart rate were affected immediately after injection of lidocaine/epinephrine but were not elevated over the normal range.[78]

3. Haemostatic Biomaterials.

Low-flow bleeding (capillary, venous, and small arteries) can be inhibited by the topical application of absorbable biomaterials. Recent development of numerous biomaterials has provided new methods for effective intraoperative and postoperative haemostasis, while avoiding complications, such as adhesions, excessive granulation tissue, and crusting.[69]

4. Topical Antifibrinolytics

Topical antifibrinolytics (i.e., epsilon-aminocaproic acid, tranexamic acid) mechanism of action is competitive binding with the lysine site on plasminogen. This prevents fibrinolysis and stabilizes the blood clot potentially decreasing further bleeding. However, the epsilon-aminocaproic acid was demonstrated to be

ineffective in reducing intraoperative bleeding. Low dose (100 mg) of tranexamic acid provided haemostasis and improved quality of the surgical field after application. [79]

5. Gelatin-Thrombin Matrix

Topical matrix sealant consists of human thrombin and gelatin matrix granules of bovine or porcine gelatin. It provides tamponade of injured vessels and rapid clot formation on the tissue surface. Topical gelatin-thrombin matrixes have been modified to allow their use during endoscopic endonasal skull base surgery. It stops bleeding on an average of 2 minutes (range 1–5 minutes) after its application. A recent study showed that bovine gelatin could effectively stop bleeding from the venous sinus. but has adverse effects such as extensive loss of cilia on the epithelium, significant increase of adhesion ($P < 0.05$) and granulation tissue formation ($P < 0.05$).[80,81,82]

6. Micro porous Polysaccharide Hemispheres

Micro porous polysaccharide hemispheres are a novel Haemostatic biomaterial agent produced from purified potato starch that acts to dehydrate blood and concentrate blood components, including platelets, red blood cells, and clotting factors. Adverse effects, including synechiae formation, oedema and infection.

Other biomaterial agents such as oxidized methyl cellulose, fibrin glue, microfibrillar collagen, and gelatin sponges can be used to control intra-operative bleeding. However, there is a lack of scientific evidence comparing the efficacy of these agents.[80]

7. Hot Water Irrigation

Hot water irrigation was originally introduced as a treatment of epistaxis. The Haemostatic mechanism of hot water irrigation is unclear but may include

i. Oedema and narrowing of the intranasal lumen that contributes to the compression of the leaking vessel
ii. Decreasing the flow and the intraluminal blood pressure due to mucosal vasodilatation; and
iii. Cleaning of blood coagulates from the nose.[83]

Hot water irrigation for epistaxis is simple and effective, less painful, and less traumatic to the nose than nasal packing therefore, this technique was adopted to reduce intraoperative bleeding. Hot water irrigation with 40^{o} –42^{o} saline reduces diffuse oozing from sinonasal mucosa as well as intracranial bleeding from minor vessels. Another benefit of warm water irrigation is that it allows the cleaning of the endoscopic lens.[69]

MATERIALS AND METHODS

A total of 50 patients undergoing bilateral endoscopic nasal surgery admitted under Department of Otorhinolaryngology and Head and Neck Surgery of R L JALAPPA HOSPITAL AND RESEARCH CENTRE, TAMAKA, KOLAR from December 2017 till June 2019 were included in the study. After proper counselling regarding the procedure being performed and the research being done written informed consent from the patients willing for surgery was taken.

Each patient received greater palatine block on one side and sphenopalatine block on another side with 2 ml of Inj 2% Lignocaine and 1:100000 adrenaline about 10 mins before starting the procedure on the respective side. Operating surgeon was blinded as to which type of block was given to which side as the blocks were administered by the assistant. All the surgeries were done by the same surgeon.

For greater palatine block:

A standard 26gauge 11/2 inch needle bent 45degrees at 25mm from tip was used under sterile precautions. [84]

Greater palatine block was given trans oral using Mercuri technique after identifying greater palatine foramen. [85]

For sphenopalatine block:

Sphenopalatine block was given trans nasal using a 25 gauge spinal needle bent 45degrees at 25mm from tip under sterile precautions.

Surgical field assessment was done every 15 minutes to assess the bleeding and was graded using Boezart and Van der Merve endoscopic grading of nasal bleeding scale.[65] Other parameters like mean arterial pressure, Heart rate, blood pressure were recorded and maintained constant

Sample size: 50 patients were included into the study

Type of study design: Prospective comparative study

Table 6: Boezaart and Van Der Merve Endoscopic Grading of Nasal Bleeding[65]

Grade	Endoscopic Grading of Nasal Bleeding-
0	No bleeding (cadaveric conditions)
1	Slight bleeding—no suctioning required
2	Slight bleeding—occasional suctioning required
3	Slight bleeding—frequent suctioning required; bleeding threatens surgical field a few seconds after.
4	Moderate bleeding—frequent suctioning required, and bleeding threatens surgical field directly after
5	Severe bleeding—constant suctioning required; bleeding appears faster than it can be removed by suction is removed suction is removed suction; surgical filed severely threatened and surgery usually is not possible

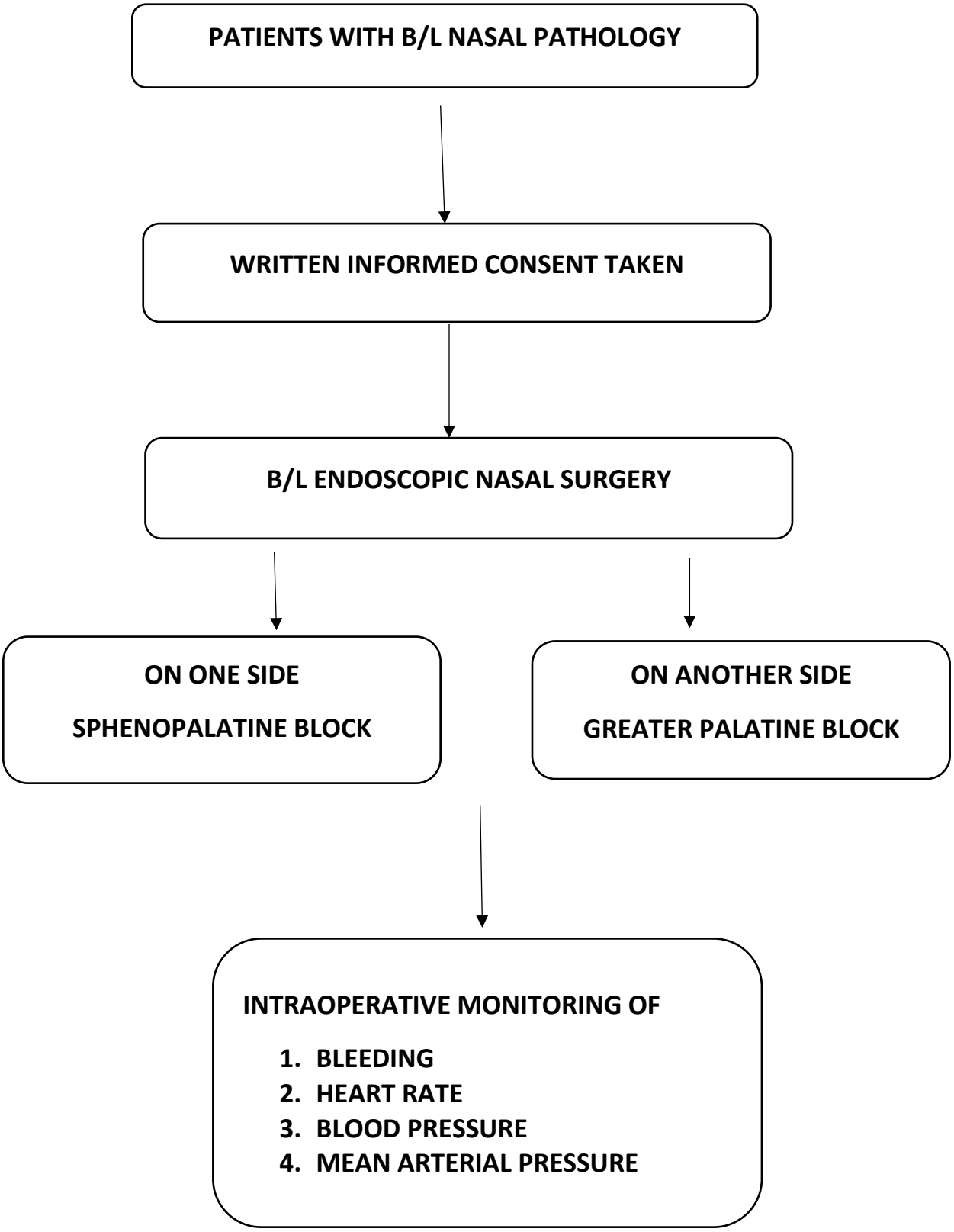


Figure 13: Study protocol

Inclusion criteria:

Patients undergoing bilateral endoscopic nasal surgery with or without DNS for

1. Bilateral Nasal polyp
2. Refractory cases of allergic rhinitis undergoing Bilateral inferior turbinoplasty
3. Chronic Rhinosinusitis

Exclusion criteria:

1. Atrophic rhinitis
2. Anatomical abnormalities
3. Malignancy of nose and PNS
4. Poorly controlled hypertension
5. Bleeding disorders
6. Use of anticoagulants

Method of collection of data:

Cases selected for the study were subjected to a detailed clinical history and complete ENT examination. All patients underwent diagnostic nasal endoscopy preoperatively to confirm the diagnosis and was graded based on Modified Lund and Kennedy system.[86] Disease were also staged based on CT imaging using the Lund and Mackay system.[87]

Table 7:The Lund and Mackay Staging System: CT Appearance score[87]

SINUS SYSTEM	LEFT	RIGHT
Maxillary (0/1/2)		
Anterior Ethmoids (0/1/2)		
Posterior Ethmoids (0/1/2)		
Sphenoid (0/1/2)		
Frontal (0/1/2)		
Osteomeatal complex (0 or 2)		
Total score		

0, No abnormalities; **1,** Partial opacification**; 2,** Total opacification.

0, Not occluded**; 2,** Occluded.

Table 8: Modified Lund and Kennedy staging system: endoscopic appearance score[86]

Characteristic		Right	Left
Polyp	(0 ,1,2)		
Oedema	(0 ,1,2)		
Discharge	(0 ,1,2)		

Polyps: 0 - Absence of polyps; 1- Polyps in middle meatus only; 2-Polyps beyond the middle meatus

Oedema: 0- Absent; 1- Mild; 2- Severe.

Discharge: 0- No discharge; 1- Clear, thin discharge; 2- Thick purulent discharge.

Cases were investigated in the following manner:

1) Haemoglobin, Total leucocyte count, Differential leucocyte count, bleeding time, Clotting time, Blood grouping and Rh typing

2) Urine for sugar, Albumin and Microscopy

3) Chest x-ray

4) ECG

5) Plain CT scan of paranasal sinuses

A correlation was established between clinical features and radiological findings. After complete pre-operative assessment, patients were subjected to surgical intervention.

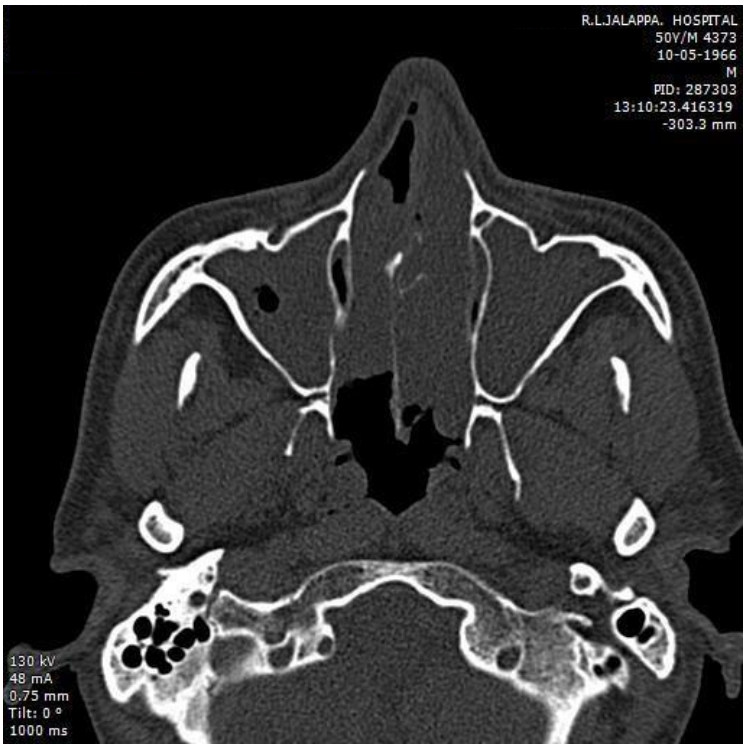


Figure 14: CT-Axial section showing bilateral maxillary sinus polyp

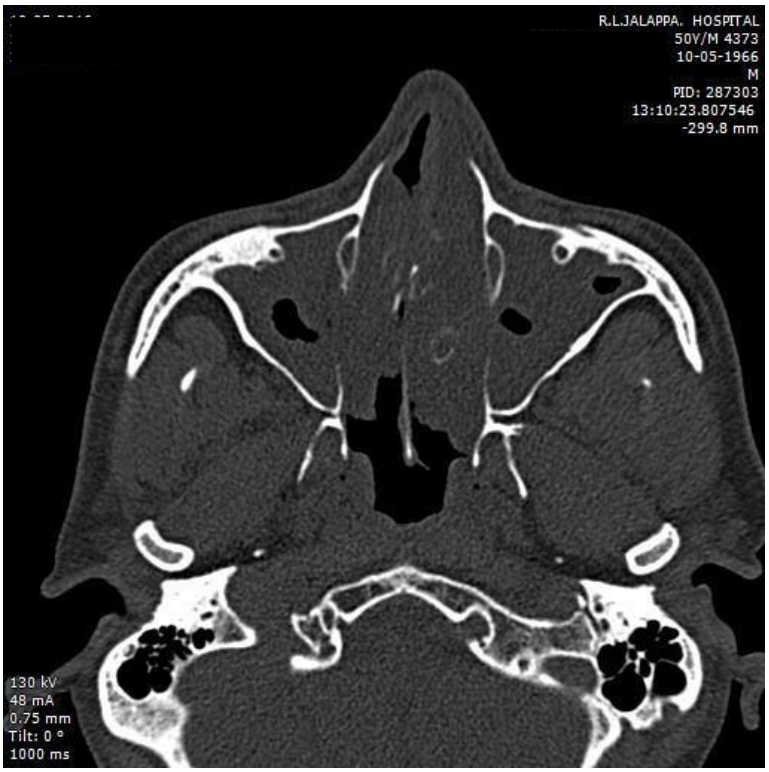


Figure 15: CT-Axial section showing bilateral osteomeatal complex occlusion

PROCEDURE

INJECTION TECHNIQUE

GREATER PALATINE BLOCK

The greater palatine foramen is located over the hard palate just anterior to the posterior edge of the hard palate opposite the second molar tooth, halfway between the tooth and the midline of the hard palate.

The greater palatine foramen was identified by palpating the palate with a finger, after depressing the tongue using a tongue depressor. An endoscope was used for visualizing. The posterior free edge of the hard palate was first palpated and then moved anteriorly over this ridge onto the hard palate. The foramen can be felt as a depression directly anterior to the free edge about midway between the second molar tooth and the midline of the palate

This region on the hard palate is identified on the monitor as the finger is withdrawn from the mouth.

The needle used for infiltration was bent at 25 mm from the tip at an angle of 45 degrees in order to perform an effective infiltration of the pterygopalatine fossa. This enables the tip of the needle to just penetrate the pterygopalatine fossa without putting any of its contents at risk (The opening of the foramen into the canal is funnel shaped and the canal is angled at 45 degrees to the hard palate). [88]

With the needle bent at 25 mm and at a 45 degree angle, the needle was inserted into the palate. If the needle struck the bone, then a small amount of was infiltrated and the needle was withdrawn assuming that the needle had just missed the foramen and that a slight adjustment was needed to locate the foramen.

If repeated attempts to identify the foramen failed, then the landmarks for the foramen were reassessed and it was relocated.

After aspirating to ensure that the needle was not in a blood vessel, the pterygopalatine fossa was infiltrated with Inj 2% Xylocaine and 1:100000 adrenaline.

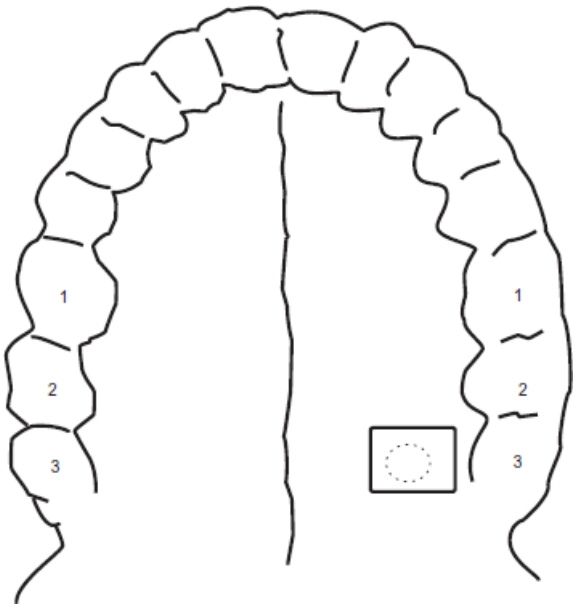


Figure 16: Schematic drawing demonstrating the position of the left greater palatine foramen in line with the third upper molar tooth on the left-hand side

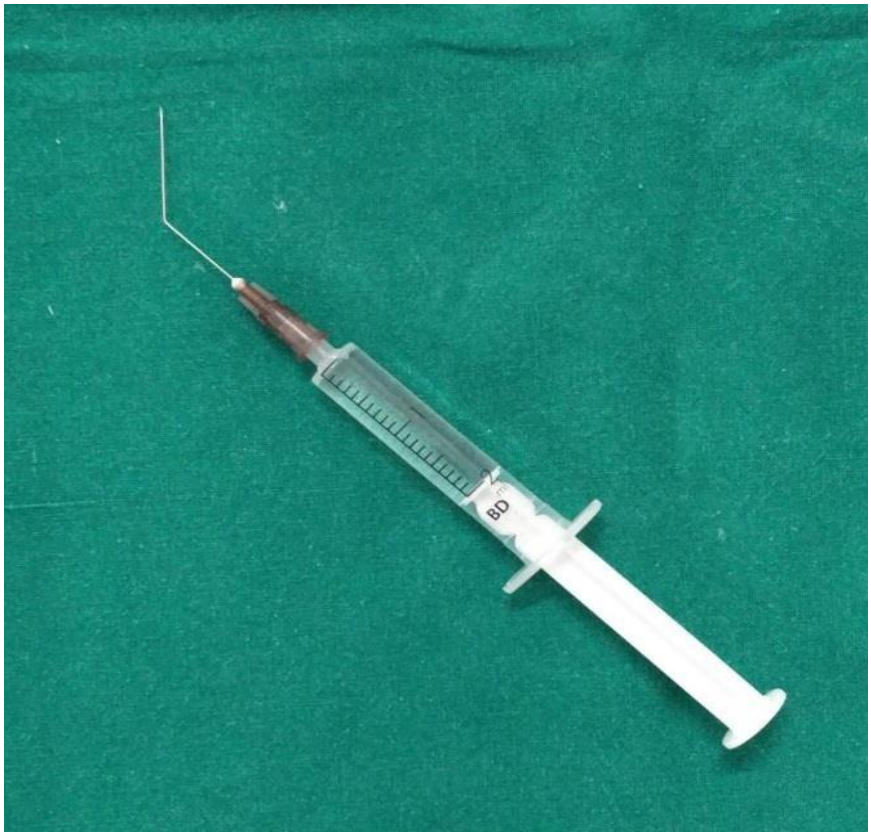


Figure 17 :26 Gauge 11/2 inch needle used to perform the greater palatine block

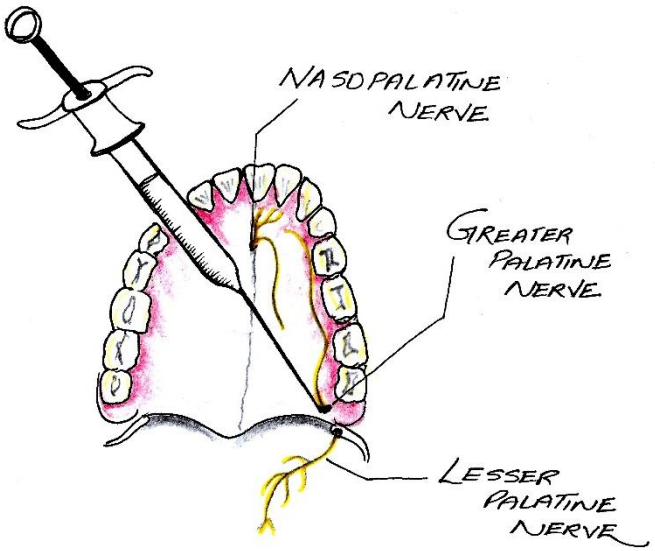


Figure 18: Schematic diagram showing transoral greater palatine block

SPHENOPALATINE BLOCK

A 25 gauge spinal needle was used. The exposed needle tip was bent 45 degrees at 25mm from the tip was used under sterile precautions. When the tip of the needle comes in contact with the posterolateral wall of the nasal cavity just posterior to the posteroinferior end of the middle turbinate, the needle was pushed into the posterolateral nasopharyngeal mucosa with the tip pointing laterally. After negative aspiration 2ml Inj 2% Lignocaine with 1:100000 Adrenaline was injected.

ANATOMICAL LOCALIZATION OF THE GANGLION

Sphenopalatine ganglion is the largest group of neurons in the head and neck. Sphenopalatine foramen which leads to sphenopalatine ganglion, lies immediately behind the ethmoidal crest situated at the posterior end of the attachment of the middle turbinate, oriented at an angle of 15-20 degrees with the sagittal plane.

Important landmark is the constant convergence of some of the vessels of lateral wall towards the sphenopalatine foramen due to the disappearance of the vessels into the foramen. This point is called the vanishing point.

The superior turbinate acts as a pointer to SPG and it is 12 mm above and lateral to the superior border of the choana. Medial to it, is the SP foramen which transmits the SP vessels and nerves from pterygopalatine fossa to the nose. The ganglion is covered by a 1-5mm layer of connective tissue and mucous membrane.

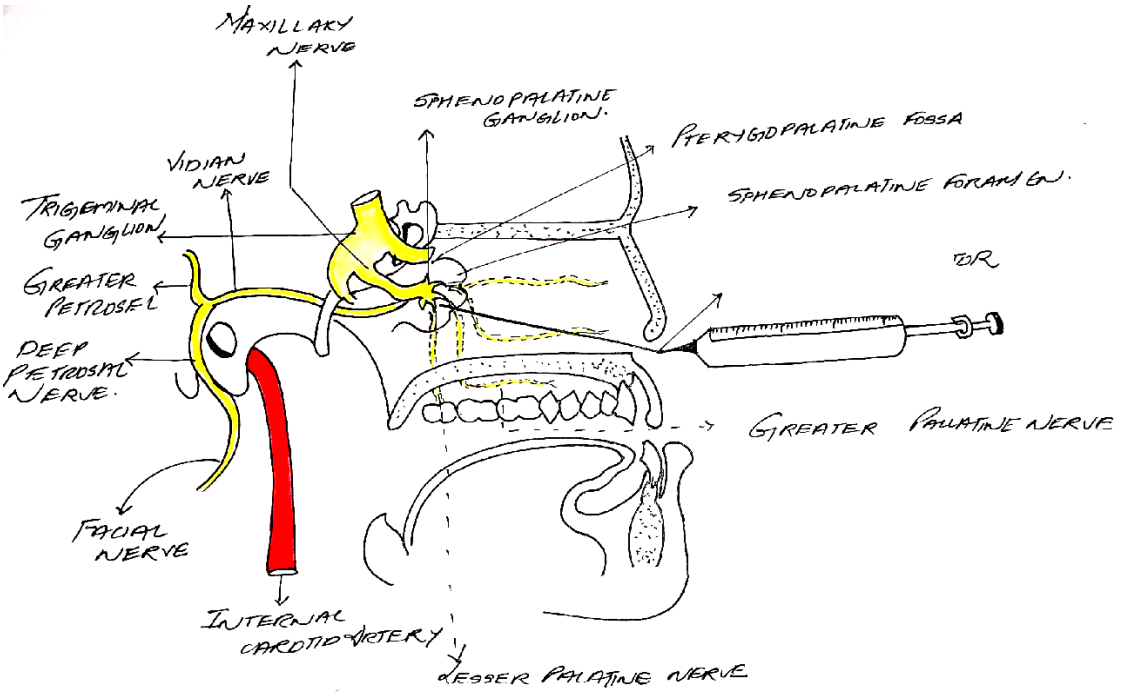


Figure 19: Schematic diagram showing transnasal sphenopalatine block

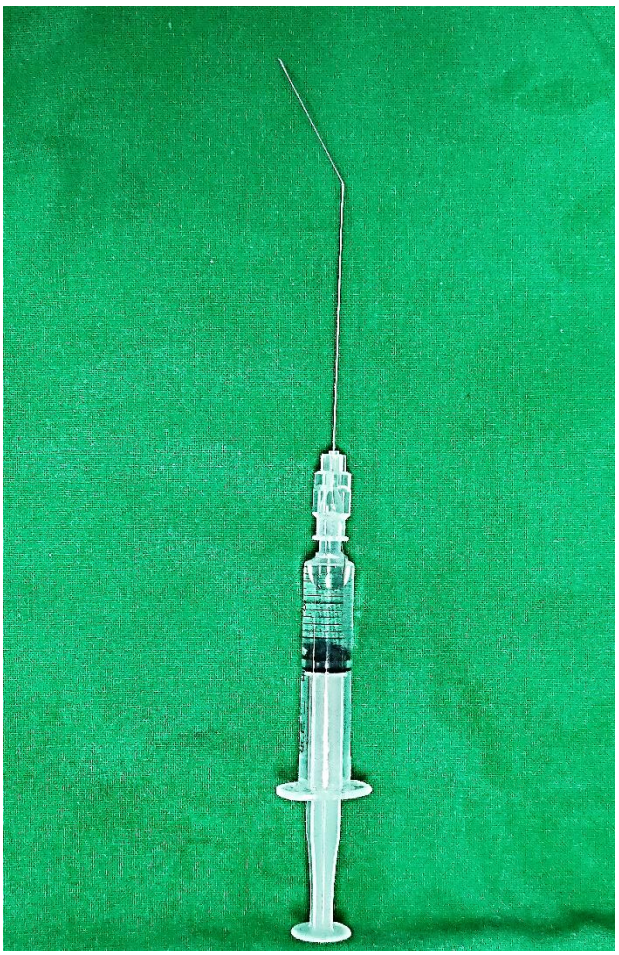


Figure 20 :25 gauge spinal needle used for sphenopalatine block

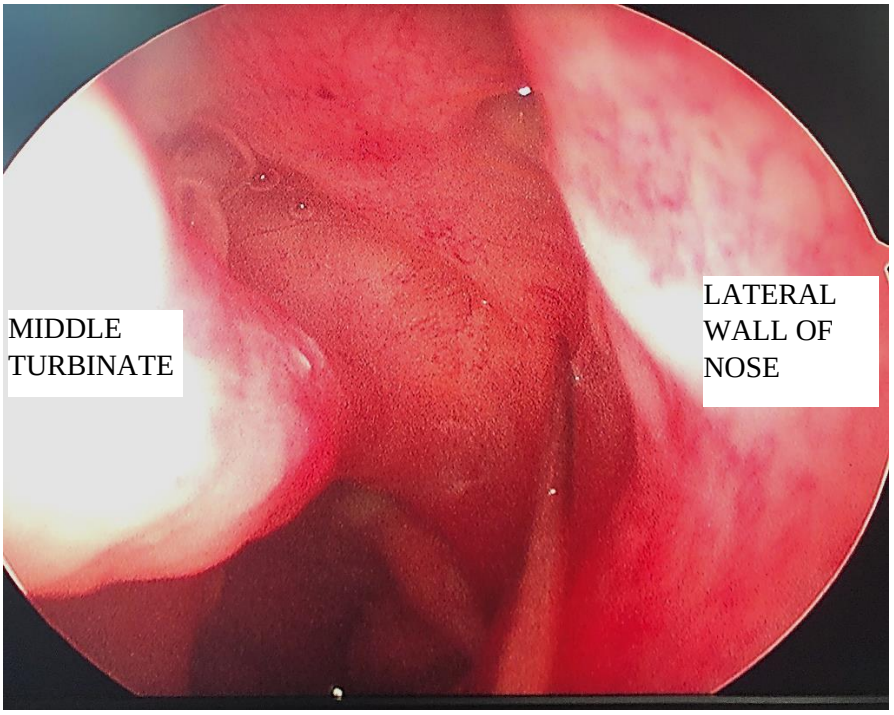


Figure 21: Endoscopic picture taken while giving sphenopalatine block

Statistical Analysis

Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. **Chi-square test or Fischer's exact test** (for 2x2 tables only) was used as test of significance for qualitative data.

Continuous data was represented as mean and standard deviation. **Independent t test** was used as test of significance to identify the mean difference between two quantitative variables

Correlations were performed with **Pearson Correlation coefficient**

Graphical representation of data: MS Excel and MS word were used to obtain various types of graphs

P value (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests.

Statistical software: MS Excel**,** SPSS version 22 **(**IBM SPSS Statistics, Somers NY, USA) was used to analyze data

RESULTS

In our study 50 patients who underwent B/L endoscopic nasal surgeries from a period of December 2017 to June 2019 were included. Each patient received greater palatine block on one side and sphenopalatine block on another side with Inj 2% Lignocaine and 1:100000 adrenaline. The effective haemostasis was assessed using "Boezaart and Van Der Merve Endoscopic Grading of Nasal Bleeding scale"

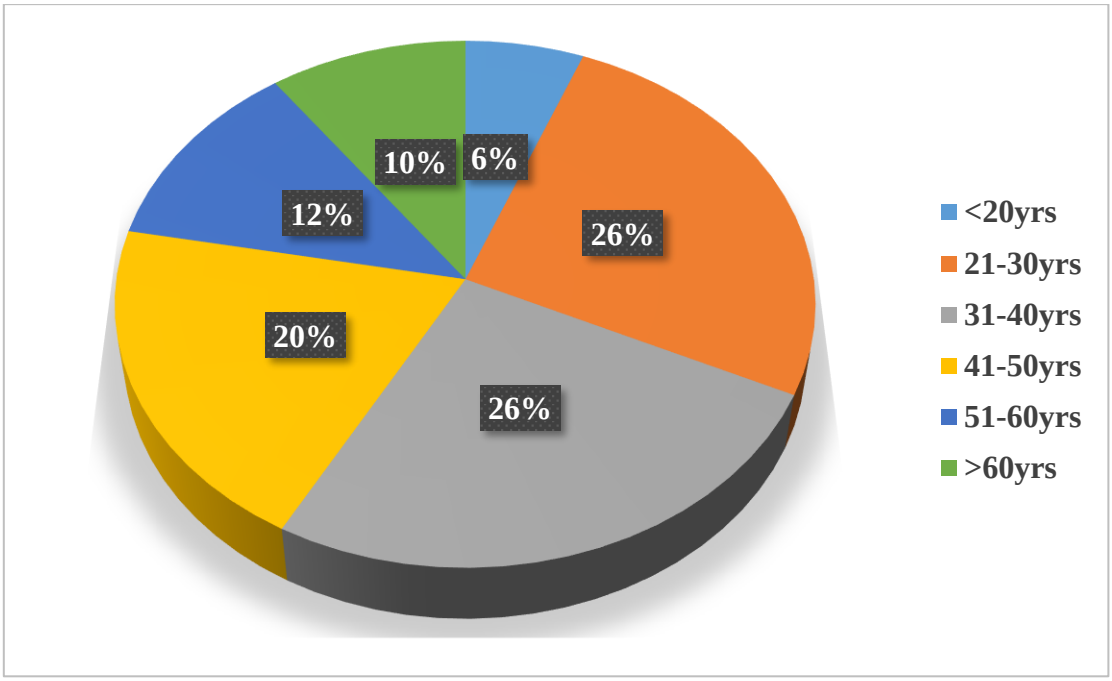
AGE DISTRIBUTION

Table 9: - Distribution of subjects according to Age group

Age Group	Frequency	Percent
<20yrs	3	6.0
21-30yrs	13	26.0
31-40yrs	13	26.0
41-50yrs	10	20.0
51-60yrs	6	12.0
>60yrs	5	10.0
Total	50	100.0

In our study Mean age 40.74 $\pm$ 15.3yrs. Minimum age 18yrs and Maximum 78yrs.

Figure 22: - Distribution of subjects according to Age group



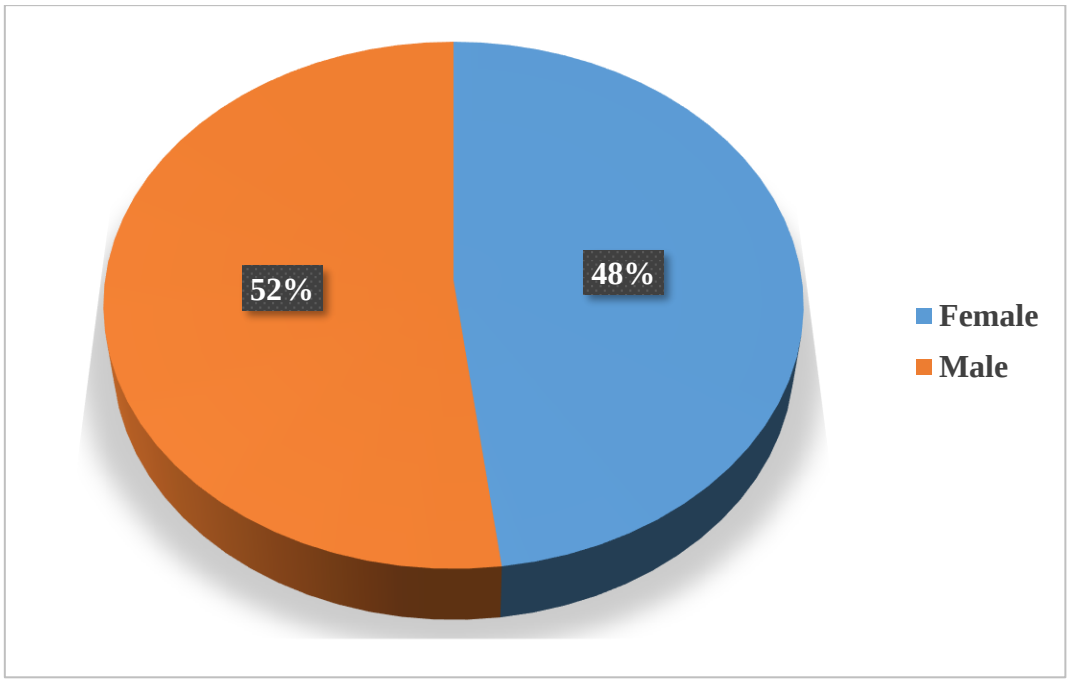
SEX DISTRIBUTION

Table 10: - Distribution of subjects according to sex

Sex	Frequency	Percent
Female	24	48.0
Male	26	52.0
Total	50	100.0

Of the 50 patients 48% were female and 52% were male.

Figure 23: - Distribution of subjects according to sex



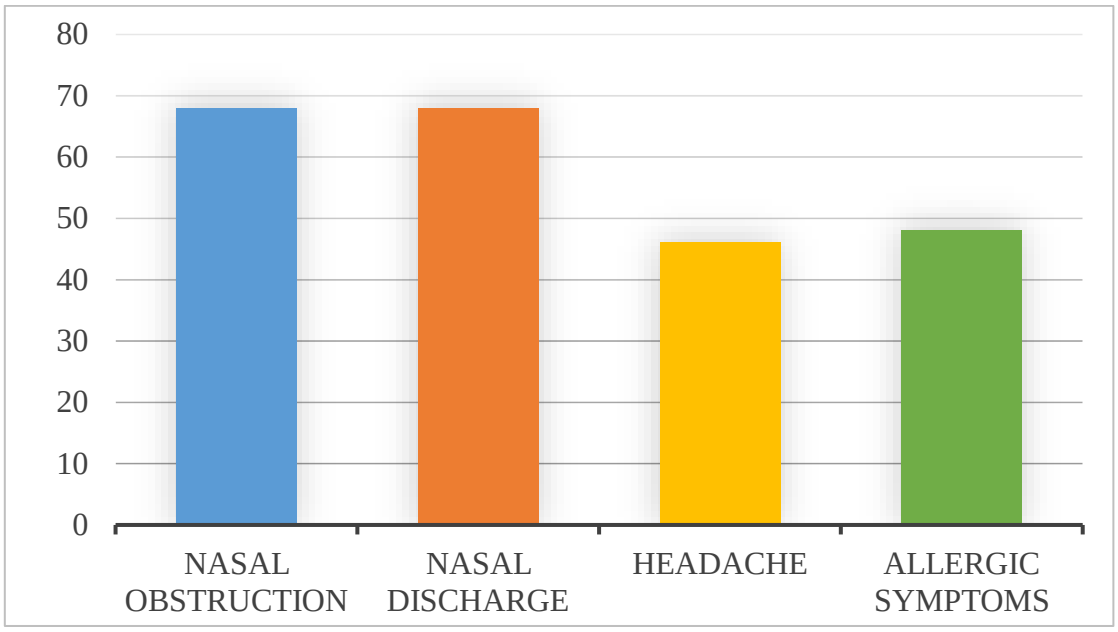
PRESENTING COMPLAINTS

Table 11: - Frequency Distribution of chief complaints

	Frequency	Percent
Nasal obstruction	34	68.0
Nasal discharge	34	68.0
Headache	23	46.0
Allergic symptoms	24	48.0

In our study 68% of the patients came with complaints of nasal obstruction, 68% with nasal discharge, 46% with headache and 48% with allergic symptoms.

Figure 24: - Bar diagram showing Frequency Distribution of chief complaints



DISTRIBUTION OF DISEASE PATHOLOGY

In our study 24% were diagnosed to have allergic rhinitis out of which 6 cases had DNS and the other 6 had no DNS , 50% with B/L ethmoidal sinonasal polyposis out of which 22 cases had no DNS and 3 cases had DNS and 26% with chronic rhinosinusitis out of which 8 cases had no DNS and 5 cases had DNS.

Figure 25: - Distribution of subjects according to diagnosis

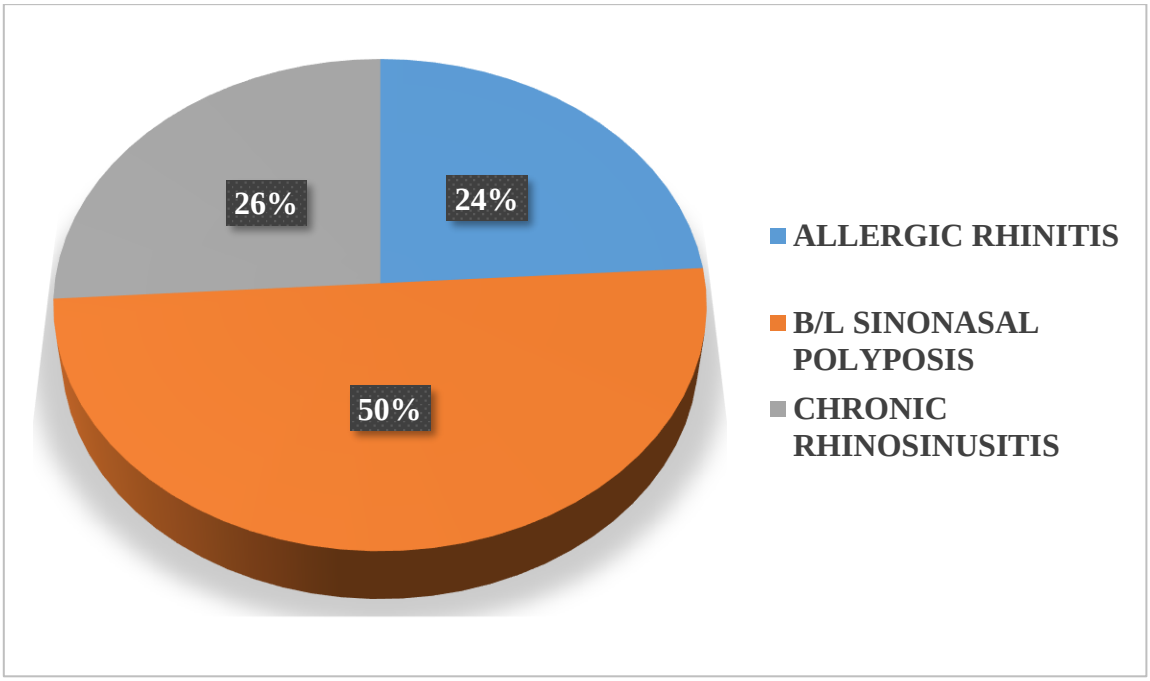
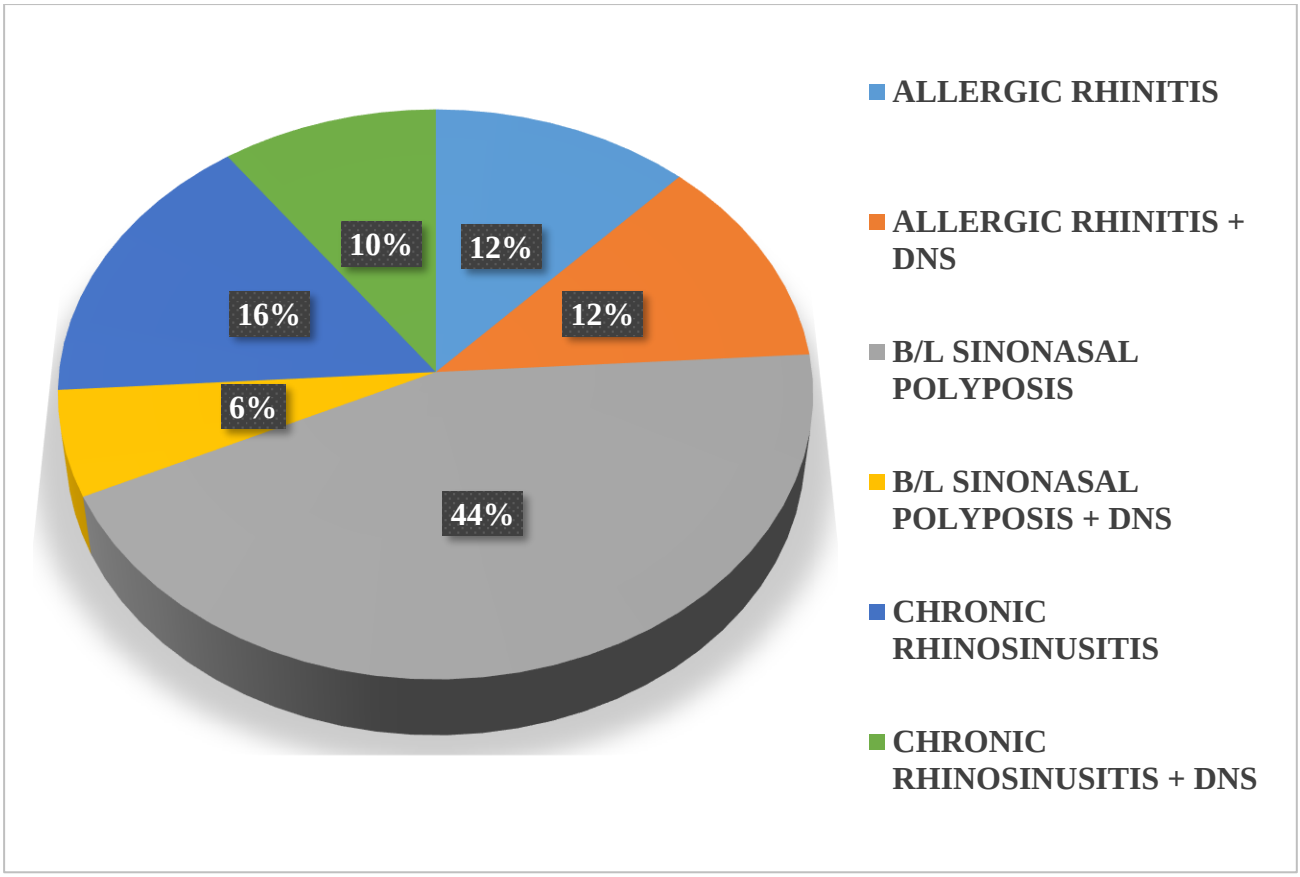


Table 12: - Distribution of subjects according to diagnosis

	Frequency	Percent
ALLERGIC RHINITIS	6	12.0
ALLERGIC RHINITIS + DNS	6	12.0
B/L SINONASAL POLYPOSIS	22	44.0
B/L SINONASAL POLYPOSIS + DNS	3	6.0
CHRONIC RHINOSINUSITIS	8	16.0
CHRONIC RHINOSINUSITIS + DNS	5	10.0
Total	50	100.0

Figure 26: - Distribution of subjects according to diagnosis



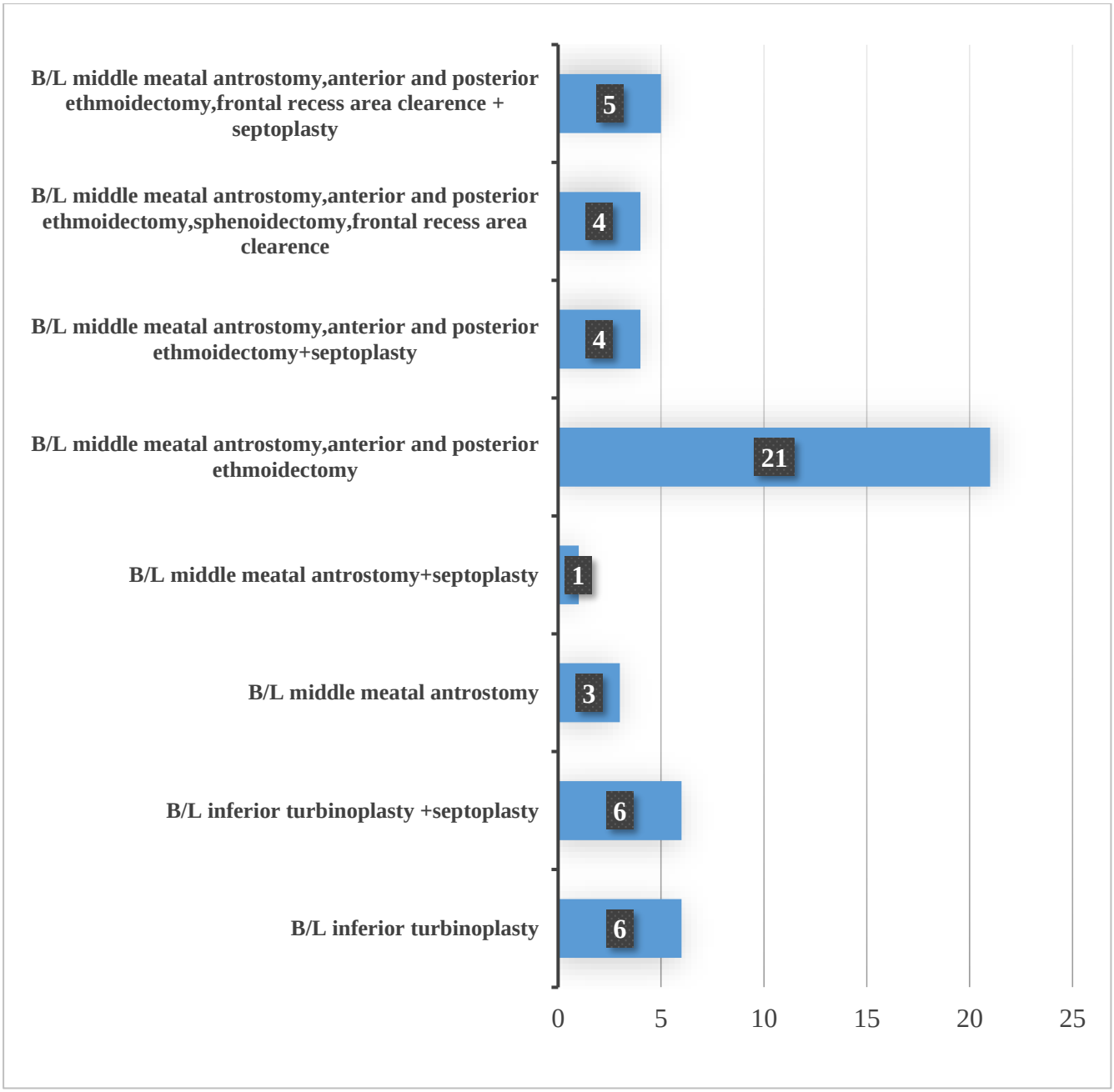
DISTRIBUTION BASED ON TYPE OF SURGERY DONE

Table 13: - Distribution of subjects according to surgery done

	Frequency	Percent
B/L inferior turbinoplasty	6	12.0
B/L inferior turbinoplasty + septoplasty	6	12.0
B/L middle meatal antrostomy	3	6.0
B/L middle meatal antrostomy + septoplasty	1	2.0
B/L middle meatal antrostomy, anterior and posterior ethmoidectomy	21	42.0
B/L middle meatal antrostomy, anterior and posterior ethmoidectomy + septoplasty	4	8.0
B/L middle meatal antrostomy, anterior and posterior ethmoidectomy, sphenoidectomy, frontal recess area clearance	4	8.0
B/L middle meatal antrostomy, anterior and posterior ethmoidectomy, frontal recess area clearance + septoplasty	5	10.0

In our study, 6 patients underwent B/L inferior turbinoplasty, 6 patients underwent ,B/L inferior turbinoplasty +septoplasty, B/L middle meatal antrostomy was done in 3, B/L middle meatal antrostomy + septoplasty in 1 patient, B/L middle meatal antrostomy, anterior and posterior ethmoidectomy 21 patients, B/L middle meatal antrostomy, anterior and posterior ethmoidectomy + septoplasty was done in 4, B/L middle meatal antrostomy, anterior and posterior ethmoidectomy, sphenoidectomy, frontal recess area clearance in 4 cases and B/L middle meatal antrostomy, anterior and posterior ethmoidectomy, frontal recess area clearance + septoplasty in 5 cases.

Figure 27: - Distribution of subjects according to surgery done



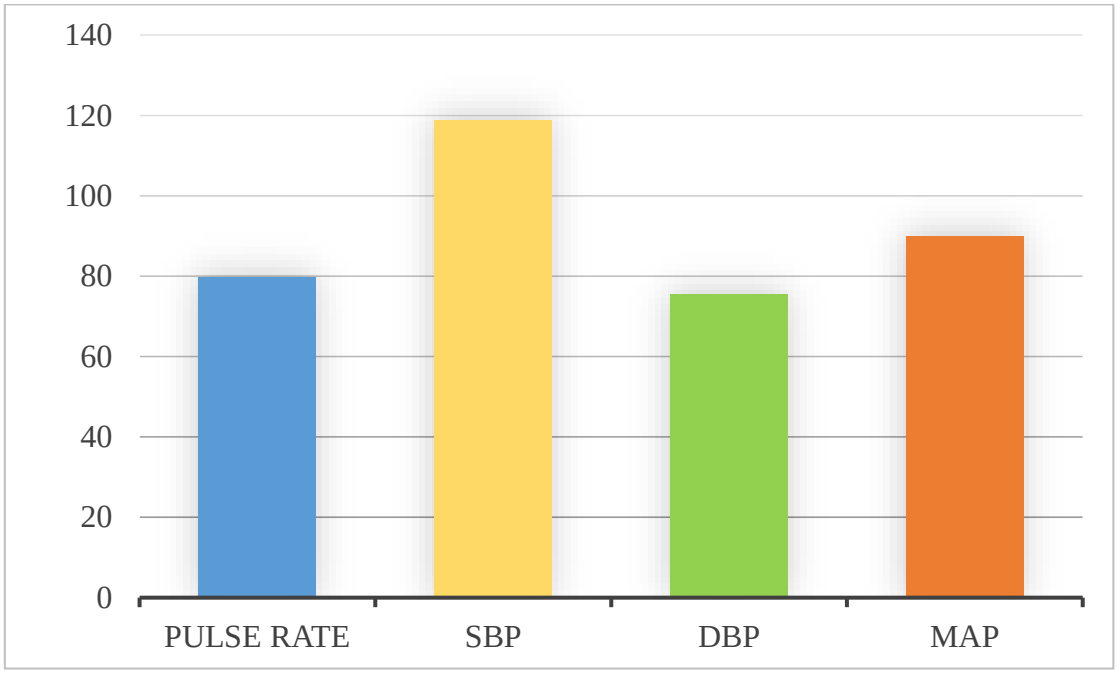
INTRAOPERATIVE PERIOD VITAL STATISTICS

Table 14: - Descriptive vital statistics during Intraoperative period

	Mean	SD
Pulse rate	79.72	2.879
Systolic blood pressure	118.76	2.454
Diastolic blood pressure	75.52	3.991
Mean arterial pressure	89.92	2.489

The mean pulse rate was 79.72± 2.879 beats/min, mean Systolic blood pressure 118.76±2.454 mmHg, mean Diastolic blood pressure 75.52±3.991mmHg and Mean arterial pressure 89.92±2.489 mmHg.

Figure 28: - Bar diagram showing Descriptive statistics of Intraoperative vitals

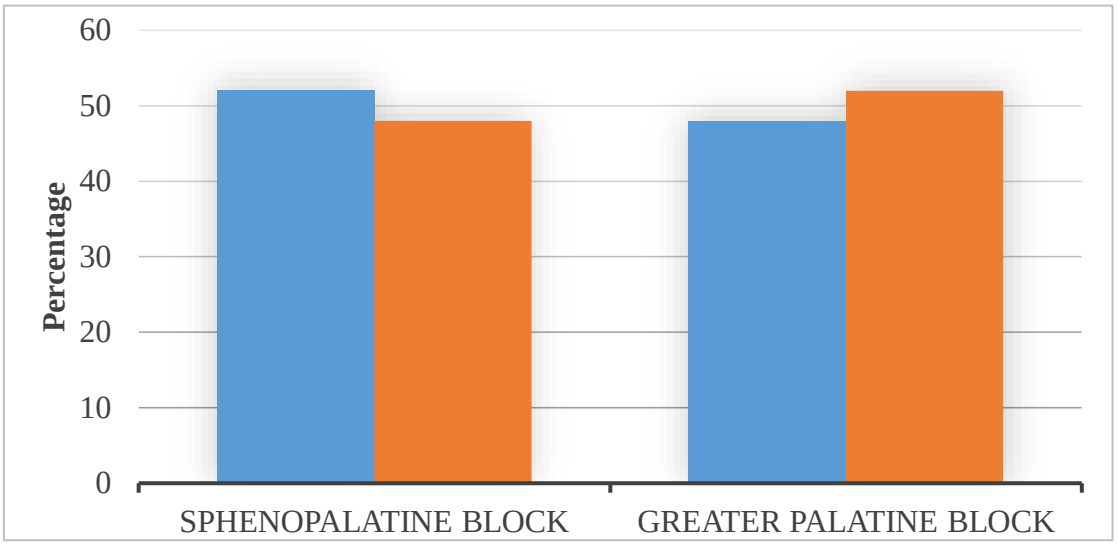


DISTRIBUTION OF BLOCK GIVEN ACCORDING TO SIDE

The sphenopalatine block was given on the right side in 24 cases and on the left side in 26 cases.

The greater palatine block was given on the right side in 26 cases and left side in 24 cases.

Figure 29: - Bar diagram showing Distribution of block given according to side



COMPARISON OF BLOOD LOSS SCORES

Table 15: - Comparison of blood loss score between sphenopalatine block and Greater palatine block according to Boezaart and Van der Merve endoscopic grading of nasal bleeding scale

	Mean	SD	P value
Sphenopalatine block	2.48250	.267273	0.287
Greater palatine block	2.42500	.301992	

In our study the mean blood loss based on the Boezaart and Van Der Merve

Endoscopic Grading of Nasal Bleeding for Sphenopalatine block was 2.48± 0.26 and Greater palatine block 2.42±0.30

Figure 30: - Bar diagram showing Comparison of blood loss score between sphenopalatine block and Greater palatine block according to Boezaart and Van der Merve endoscopic grading of nasal bleeding scale

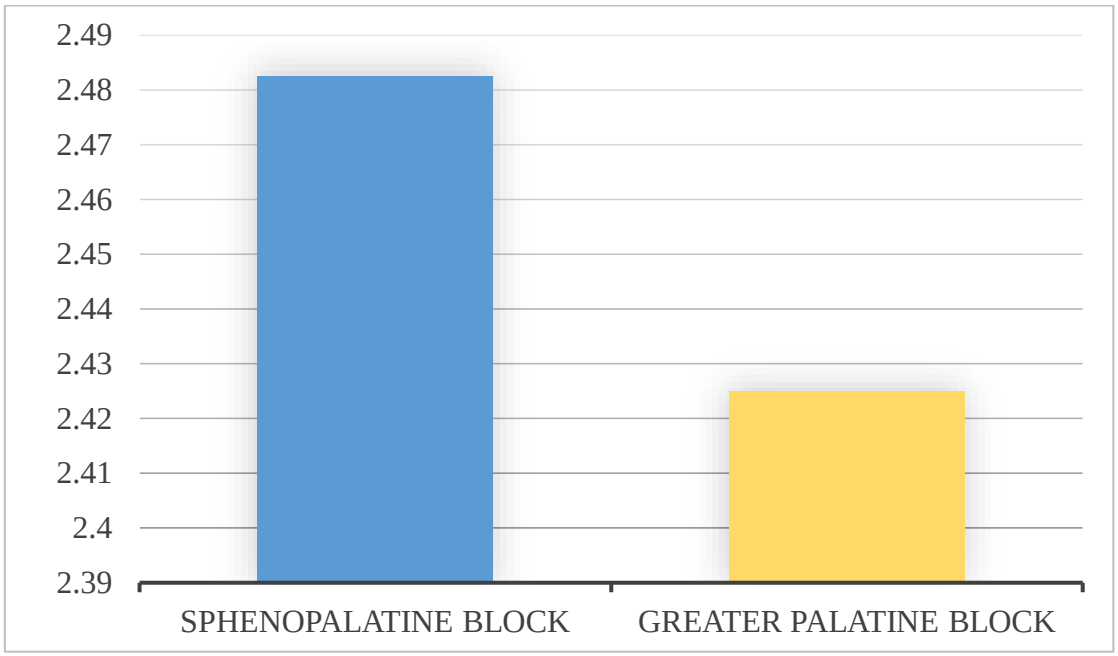


Table 16: - Comparison of sphenopalatine block and greater palatine block scores based on different surgery according to Boezaart and Van der Merve endoscopic grading of nasal bleeding scale

	Sphenopalatine block		Greater palatine block	
	Mean	SD	Mean	SD
B/L inferior turbinoplasty	2.563	.105	2.375	.316
B/L inferior turbinoplasty + septoplasty	2.521	.166	2.188	.385
B/L middle meatal antrostomy	2.375	.217	2.583	.144
B/L middle meatal antrostomy, anterior and posterior ethmoidectomy	2.440	.344	2.512	.285
B/L middle meatal antrostomy, anterior and posterior ethmoidectomy, frontal recess area clearance + septoplasty	2.575	.244	2.325	.371
B/L middle meatal antrostomy, anterior and posterior ethmoidectomy, sphenoidectomy, frontal recess area clearance	2.531	.213	2.313	.239
B/L middle meatal antrostomy, anterior and posterior ethmoidectomy + septoplasty	2.438	.315	2.438	.125
B/L middle meatal antrostomy + septoplasty	2.500	.	2.750	.

The mean blood loss score for sphenopalatine block was maximum 2.575± 0.244 and seen in B/L middle meatal antrostomy, anterior and posterior ethmoidectomy, frontal recess area clearance + septoplasty surgery; and minimum 2.440±0.344 seen in B/L middle meatal antrostomy, anterior and posterior ethmoidectomy.

The mean blood loss score for greater palatine block was maximum 2.750 and seen in B/L middle meatal antrostomy surgery; and minimum 2.188±0.385 seen in B/L inferior turbinoplasty + septoplasty.

CORRELATION OF CT SCAN GRADE, ENDOSCOPIC GRADE AND BLOOD LOSS SCORE IN SPHENOPALATINE BLOCK

Table 17: - Correlation of CT scan grade and Endoscopic grading with blood loss score in Sphenopalatine block

		blood loss grade
CT Scan Grade	Pearson Correlation	.219
	P value	.186
	N	38
Endoscopic grading	Pearson Correlation	-.043
	P value	.795
	N	38

Table 18: - Correlation of CT scan grade and Endoscopic grading with blood loss grade in Greater palatine block

		Blood loss grade
CT Scan Grade	Pearson Correlation	-.072
	P value	.669
	N	38
Endoscopic grading	Pearson Correlation	.137
	P value	.412
	N	38

In our study no statistically, significant correlation was observed between CT scan grade based on Lund and Mackay CT scan appearance score, endoscopic grade based on Modified Lund and Kennedy score and blood loss based on Boezaart and Van der Merve endoscopic grading of nasal bleeding score in both sphenopalatine block and greater palatine block.

CORRELATION OF INTRAOPERATIVE VITAL STATISICS WITH SPHENOPALATINE AND GREATER PALATINE BLOCK

Table 19: - Correlation of vitals sign with blood loss score according to Boezaart and Van der Merve endoscopic grading of nasal bleeding in Sphenopalatine block and in Greater palatine block

		Sphenopalatine block	Greater palatine block
		Blood loss grade	Blood loss grade
Pulse rate	Pearson Correlation	0.033	.087
	P value	.818	.549
	N	50	50
Systolic blood pressure	Pearson Correlation	-0.158	.175
	P value	.272	.225
	N	50	50
Diastolic blood pressure	Pearson Correlation	-0.137	-.047
	P value	.342	.744
	N	50	50
Mean arterial pressure	Pearson Correlation	-0.202	.005
	P value	.161	.970
	N	50	50

In our study both in sphenopalatine block and greater palatine block there was no statistically significant correlation observed between pulse rate, SBP, DBP and MAP with blood loss score.

DISCUSSION

Endoscopic surgery targeted at the paranasal sinuses and the turbinates has become very popular in the present era and is being done at majority of the hospitals involved in ENT practice. Due to the highly vascular nature of nasal tissue, haemostasis has always been a concern. Visualization of the nasal anatomy is vital to the procedure, allowing complete dissection, and avoiding complications. Bleeding from the nasal mucosa during ESS interferes with the surgical field, prolongs operative time, and increases the incidence of incomplete surgery. Hence, most surgeons agree that haemostasis is of paramount importance.

In our study most of the patients were adults in the age group of 21-40 years. The mean age of the subjects in our study was 40.74 ± 15.3yrs with minimum age 18yrs and maximum 78yrs.Similarly in a study conducted by Wormald et al the median age was 50 years (range , 20-78 years).[1] In another study conducted it was found that majority of the patients who presented were also in the age group of 16 to 45 years.[89] this could be due to the high prevalence of allergic rhinitis and rhinosinusitis in this region particularly among the working class majority of whom are manual labourers or farmers exposed to dust.

There was no gender predisposition with 26 (52%) male patients and 24 (48%) females. This is because both males and females are involved in manual labour in this region and seek medical help only when the condition is chronic. In all other studies that were compared males were predominant in number.[1]

The presenting symptoms were nasal obstruction in 68% of cases, nasal discharge in 68%, headache in 46% cases and allergic symptoms in 48% cases.

Of the 50 patients, 50% of subjects were diagnosed to have B/L sinonasal polyposis, 26% had chronic rhinosinusitis and 24% had allergic rhinitis. In another study

conducted the most common condition was chronic rhinosinusitis (50%) and the remaining 50 % were diagnosed as polyposis and fungal sinusitis.[1] We had higher number of patients with sinonasal polyposis because our hospital is in an economically backward region and patients seek medical help only when the health problem is long standing or severe.

Majority of the patients in our study underwent B/L middle meatal antrostomy, anterior and posterior ethmoidectomy i.e. 21 patients as they were found to have pansinusitis. this could be due to obstruction of the sinus drainage due to polypi. Other studies in literature have also reported that almost 40-50% of their patients underwent endoscopic sinus surgery for nasal polypi.[1,7,90] 12 patients underwent B/L inferior turbinoplasty with or without septoplasty for B/L inferior turbinate hypertrophy; this was because they also had allergic rhinitis.

In our study the Mean Heart rate was 79.72 bpm, SBP was 118.76 mmHg, DBP was 75.52 mmHg and MAP was 89.92 mmHg. In our study however no significant correlation was observed between blood loss score according to Boezaart and Van der Merve endoscopic grading of nasal bleeding scale and heart rate, SBP, DBP and MAP respectively with both greater palatine and sphenopalatine block. In a study done by Wormald et al. they have not found any statistically significant correlation between MAP and surgical grade with greater palatine block (p value = 0.724).[1]

In a similar study done by Sieśkiewicz A et al, they concluded that significant correlation was present between mean arterial pressure and conditions in the operating field (p value =0.003). They attained good operative field with a mean arterial pressure in the range of 65-78 mm Hg. In this study no correlation was found between heart rate and bleeding in the operating field as the authors had maintained heart rate in the range of 60 / min.

In our study the mean blood loss score for every 15 mins on sphenopalatine block side was 2.48 ± 0.26 and on greater palatine block side was 2.42 ± 0.30. P =0.287. This showed that there was no significant difference in haemostasis between greater palatine block and sphenopalatine block. The reason for this maybe that both the above-mentioned blocks help to cause vasoconstriction of both sphenopalatine and greater palatine arteries by acting on post ganglionic nerve fibres in pterygopalatine fossa. However, with both the above mentioned blocks haemostasis was fairly good and the score according to Boezaart and Van der Merve endoscopic grading of nasal bleeding scale sphenopalatine block side was 2.48 ± 0.26 and on greater palatine block side was 2.42 ± 0.30 showing that there was slight bleeding and only required occasional suctioning to provide good visualisation of the field.

There have been numerous studies comparing effect of pterygopalatine fossa injection via the greater palatine foramen to evaluate its effect and estimate blood loss during endoscopic sinus surgery.

Wormald et al, conducted a study in 2005 which showed statistically significant benefit in favour of the pterygopalatine fossa injection when comparing endoscopic surgical field. The overall mean blood loss grade of the injected site was 2.59 when compared to 2.99 for the non-injected site.[1]

Other studies where the blood loss was calculated by points of ooze based on Wormald surgical field grading scale, have also reported decreased bleeding on the side of greater palatine block when compared to the other side where no block was given.[91]

In another study conducted by Valdes et al they found no statistical difference between injected and non-injected site in terms of surgical grade and blood loss. Furthermore, there was no correlation between surgical field improvement, MAP,

heart rate either.[92]

In a study conducted by Salah A. Ismail they concluded that bilateral sphenopalatine ganglion block via the greater palatine fossa provided a satisfactory operative field with less blood loss.[93]

There were no complications while giving either of these blocks in our study. In other studies the authors have reported no major complications related to the injection technique of greater palatine block.[1,7,90] In one study minor postoperative complaints like dental numbness and transient sense of retro-ocular pressure were seen on the side of giving block.[93]

This shows that when administered carefully both greater palatine block and sphenopalatine block are safe and provide good haemostasis for nasal and endoscopic paranasal sinus surgeries.

However almost all studies documenting the effect of pterygopalatine fossa block for nasal and endoscopic sinus surgeries had relatively small sample size ranging between 40-55 patients each.[1,7,90] Having seen the encouraging results in our study as well as in literature, it would be desirable to have a multi institutional study with a large sample size to study the effect of pterygopalatine fossa block on bleeding in nasal and endoscopic sinus surgeries. If similar results as ours are found in such a large study, pterygopalatine fossa block can be recommended as part of a regular protocol for nasal and endoscopic sinus surgeries.

SUMMARY

50 Patients undergoing bilateral endoscopic nasal surgery admitted under Department of Otorhinolaryngology and Head and Neck Surgery of R L JALAPPA HOSPITAL AND RESEARCH CENTRE, TAMAKA, KOLAR from December 2017 till June 2019 were included in the study

- Maximum incidence was seen in the 3rd and 4th decade of life.
- Patients undergoing bilateral endoscopic nasal surgery for Bilateral Nasal polyp, Refractory cases of allergic rhinitis undergoing Bilateral inferior turbinoplasty, Chronic Rhinosinusitis were included.
- Patients with atrophic rhinitis, anatomical abnormalities, malignancy of nose and PNS, poorly controlled hypertension, bleeding disorders and patient on anticoagulants were excluded.
- 50% of the cases were diagnosed to have B/L sinonasal polyposis;
- Majority i.e. 21 patients underwent B/L middle meatal antrostomy, anterior and posterior ethmoidectomy.
- All patients underwent endoscopic nasal surgery following injection of the drug Inj 2% lignocaine with 1:100 either by transoral greater palatine block and with the transnasal sphenopalatine block. Intra-operatively surgical field assessment was done every 15 minutes to assess the bleeding and was graded using Boezart and Van der Merve endoscopic grading of nasal bleeding scale.
- Mean Blood loss grade for sphenopalatine block was 2.48± 0.26 and for greater palatine block was 2.42 ± 0.30 which showed there was no significance difference between the 2 blocks with regard to haemostasis because both the blocks acted on the post ganglionic fibres of the

pterygopalatine ganglion bringing about vasoconstriction of sphenopalatine and greater palatine arteries. However, both the blocks gave fairly good haemostasis as indicated in the bleeding score giving good visualisation of the surgical field. Similar results have been reported by various studies which have used greater palatine block for nasal and endoscopic sinus surgeries.[1,7,90,94]

- No significant correlation was found between the sphenopalatine and greater palatine blocks and intraoperative heart rate, SBP, DBP and MAP. Similarly majority studies in literature have reported no correlation. [1,7]
- No complications were encountered during the procedure.

CONCLUSION

Achieving a blood less field during endoscopic nasal and paranasal sinus surgery remains a challenge to date.

Both greater palatine block and sphenopalatine block provided fairly good haemostasis facilitating good visualisation of the surgical field and requiring occasional suctioning.

Mean Blood loss grade for sphenopalatine block was 2.48± 0.26 and for greater palatine block was 2.42 ± 0.30 on Boezaart and Van der Merve endoscopic grading of nasal bleeding scale.

There was no significant difference in haemostasis between sphenopalatine block and greater palatine block in our study.

However greater palatine block was easier to administer since it was transoral and identifying the greater palatine foramen is relatively easy.

Furthermore, it was found that the grade of bleeding was not dependent on various factors like mean arterial pressure, blood pressure and heart rate.

When administered carefully both the blocks are safe and no complications was encountered.

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ANNEXURES

PROFORMA

COMPARISON OF SPHENOPALATINE AND GREATER PALATINE BLOCK IN MINIMISING BLEEDING DURING ENDOSCOPIC NASAL SURGERY

PERSONAL DETAIL

Name: Age: Sex:

Occupation: Hospital no:

Address:

PRESENTING COMPLAINT

CHIEF COMPLAINTS	YES/NO	SINCE
NASAL OBSTRUCTION		
NASAL DISCHARGE		
HEADACHE		
ALLERGIC SYMPTOMS		

HISTORY OF PRESENT ILLNESS

PAST HISTORY

COMORBIDITIES	YES/NO	SINCE
Hypertension		
Diabetes Mellitus		

Pulmonary Tuberculosis		
Bronchial Asthma		

H/O drug allergy: Y/N
H/O previous surgery: Y/ N

Treatment History (if any): Surgery

FAMILY HISTORY

Contributory ☐ Not contributory ☐

PERSONAL HISTORY

Loss of appetite: Y/ N Bowel and bladder disturbances: Y/ N

Disturbed sleep: Y/ N Habits –

EXAMINATION

GENERAL PHYSICAL EXAMINATION

Built: Poor ☐
Medium ☐
Well-built ☐

Nutritional status: Poor ☐
Satisfactory ☐

Temperature: Pulse: BP: RR:

Pallor: Y/ N Icterus: Y/ N Cyanosis Y/ N

Clubbing: Y/ N Lymphadenopathy: Y/ N Edema: Y/ N

LOCAL EXAMINATION

- **Nose:**

  External framework

  Vestibule

  Columella

  Anterior Rhinoscopy
  Nasal cavity-
  Septum-

Mucosa-
Turbinate-
Floor of nose-

Posterior Rhinoscopy

PNS tenderness

DNE finding

	Right side	**Left side**
1st pass		
2nd pass		
3rd pass		

- **Oral Cavity:**

  Mouth opening: Adequate/ Trismus

  Oro-dental Hygiene: Poor/ Satisfactory

  Lips

  Teeth

  Tongue

- **Oropharynx**

  AP

  PP

  PPW

- **Ear**

  RIGHT LEFT

  Pre- and Post-auricular region

  Pinna

  EAC

TM

Facial Nerve

SYSTEMIC EXAMINATION:

- Cardio vascular system:
- Respiratory system:
- Abdomen:
- Central nervous system:

CLINICAL DIAGNOSIS :

INVESTIGATIONS :

Hb: RBC: TC: Platelets:

DC: N: L: M: E: B:

BT: CT: HIV: Y/ N HbsAg: Y/ N RBS:

CT PNS: The Lund and Mackay Staging System: CT Appearance score

SINUS SYSTEM	LEFT	RIGHT
Maxillary (0/1/2)		
Anterior Ethmoids (0/1/2)		
Posterior Ethmoids (0/1/2)		
Sphenoid (0/1/2)		
Frontal (0/1/2)		
Osteomeatal complex (0 or 2)		
Total points:		

0, No abnormalities; **1,** Partial opacification**; 2,** Total opacification.

0, Not occluded**; 2,** Occluded.

MODIFIED LUND AND KENNEDY STAGING SYSTEM: ENDOSCOPIC APPEARANCE SCORE

Characteristic	Baseline
Polyp -- > Left / Right (0 ,1,2)	
Oedema --> Left / Right (0 ,1,2)	
Discharge --> Left / Right (0 ,1,2)	

Polyps: 0, Absence of polyps; 1, Polyps in middle meatus only; 2, Polyps beyond the middle meatus

Oedema: 0, Absent; 1, Mild; 2, Severe.

Discharge: 0, No discharge; 1, Clear, thin discharge; 2, Thick purulent discharge.

OPERATIVE FINDINGS:

Surgery done:

Date of surgery:

INTRAOPERATIVE FINDINGS:

GRADING OF BLEEDING: **BOEZAART AND VAN DER MERV SCALE**

TIME	RIGHT	LEFT
0 min		
15 min		
30 min		
1 hr		
1hr 15min		
1hr 30min		
1hr 45min		
2 hr		
2hr 15 mins		

0 No bleeding (cadaveric condition)

1 Slight bleeding-No suctioning required

2 Slight Bleeding-Occasional suctioning required

3 Slight Bleeding-Frequent suctioning required and bleeding threatens surgical field a few seconds after

4 Moderate Bleeding-Frequent suctioning and bleeding threatens surgical field directly after suctioning

5 Severe bleeding-constant suctioning required and bleeding faster than it can be removed by suctioning, surgery not possible

OTHER PARAMETERS:

TIME	HR	SBP	DBP	MAP	END TIDAL CO2
0 min					
15 min					
30 min					
45 min					
1 hr					
1hr 15min					
1hr 30min					
1hr 45min					
2hr					
2hr 15min					
2hr 30min					

OUTCOME OF SURGERY:

Total score based on Boezaart and Van der Merve endoscopic grading of nasal bleeding for –

i. Greater palatine block

ii. Sphenopalatine block

PATIENT INFORMATION SHEET

STUDY TITLE: COMPARISON OF SPHENOPALATINE AND GREATER PALATINE BLOCK IN MINIMISING BLEEDING DURING ENDOSCOPIC NASAL SURGERY

STUDY LOCATION: R L Jalappa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar.

AIM: To compare the effective haemostasis with sphenopalatine and greater palatine block during endoscopic surgeries of the nose and paranasal sinuses.

STUDY DETAILS: The surgical field in endoscopic nasal surgery is very narrow and is surrounded by vital structures. Even a small amount of bleeding during surgery can soil the tip of the endoscope and obscure the field and this increases the risk of injury to the adjacent vital structures. Hence an optimal bloodless field is crucial for the surgeon. the sphenopalatine and greater palatine cause vasoconstriction of the vessels supplying the nose and hence giving better haemostasis. With this study we intend to compare the two blocks in terms of better haemostasis. The complications with these include bleeding, injury to adjacent structures. Please read the following instructions and discuss with your family members. You can ask any question regarding the study. If you agree to participate in the study, we will collect information (as per proforma) from you. A detailed clinical history will be taken and clinical examination done. Relevant blood and radiological investigations will be carried out.

All information collected from you will be kept confidential and will not be disclosed to any outsider. All information collected will only be used for dissertation and publication purposes. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the member of Institutional Ethics

Committee. You will not have any kind of financial benefits for being part of this study nor will incur any additional expenses for being a part of this study. There is no compulsion to participate in this study. The care you will get will not change if you do not wish to participate.

You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

WHO TO CONTACT?

For further information

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STUDY TITLE: COMPARISON OF SPHENOPALATINE AND GREATER PALATINE BLOCK IN MINIMIZING BLEEDING DURING ENDOSCOPIC NASAL SURGERY

INFORMED CONSENT FORM

I, Mr. / Mrs. have been explained in a language I can understand, that I will be included in a study which is "Comparison of sphenopalatine and greater palatine block in minimizing bleeding during endoscopic nasal surgery"

I have been explained that my clinical finding, investigations, intraoperative findings will be assessed and documented for the study purpose.

I have been explained that my participation in this study is entirely voluntary and I can withdraw from the study anytime and this will not affect my relation with my doctor or the treatment for my ailment.

I have understood that all my details found during the study are kept confidential and while publishing or sharing of the findings, my details will be masked.

I, willingly give my full consent to be part of this study.

Signature of the patient:

Name:

Signature of the witness:

Name:

Date: Place:

KEY TO MASTERCHART

- M- MALE
- F- FEMALE
- B/L-BILATERAL
- CT SCAN GRADE- LUND AND MACKAY CT SCAN SCORE
- ENDOSCOPIC SCORE- MODIFIED LUND AND KENNEDY ENDOSCOPIC SCORE
- MMA- MIDDLE MEATAL ANTROSTOMY
- AE-ANTERIOR ETHMOIDECTOMY
- PE-POSTERIOR ETHMOIDECTOMY
- S-SPHENOIDECTOMY
- FRC-FRONTAL RECESS AREA CLEARENCE
- IT-INFERIOR TURBINOPLASTY
- SEPTO-SEPTOPLASTY
- IOV-INTRAOPERATIVE VITALS
- HR-HEART RATE
- SBP-SYSTOLIC BLOOD PRESSURE
- DBP-DIASTOLIC BLOOD PRESSURE
- MAP-MEAN ARTERIAL PRESSURE
- SP BLOCK-SPHENOPALATINE BLOCK
- GP BLOCK-GREATER PALATINE BLOCK
- POC-POST OPERATIVE COMPLICATIONS

HOSPITAL NO.	AGE	SEX	OCCUPATION	CHIEF COMPLAINTS								COMORB	DIAGNOSIS	CT SCAN SCORE		ENDOSCOPIC SCORE		SURGERY DONE	IOV				BLEEDING SCORE					POC
				NASAL OBSTRUCTION	DURATION	NASAL DISCHARGE	DURATION	HEADACHE	DURATION	ALLERGIC SYMPTOMS	DURATION			RIGHT	LEFT	RIGHT	LEFT		HR	SBP	DBP	MAP	SP BLOCK		GP BLOCK		
														TOTAL	TOTAL	TOTAL	TOTAL						SIDE	TOTAL	SIDE	TOTAL	
533544	35	M	CLERK	P	6 MONTHS	A	~	P	2 MONTHS	A	~	NIL	B/L SINONASAL POLYPOSIS	4	6	2	2	FESS-B/L MMA,AE,PE	78	120	76	91	R	18	L	22	Nil
525543	45	F	HOMEMAKER	P	1 YEAR	P	1 MONTH	A	~	A	~	NIL	B/L SINONASAL POLYPOSIS	4	4	3	3	FESS-B/L MMA,AE,PE	80	118	80	93	R	20	L	18	Nil
536574	46	M	FARMER	P	2 YEARS	A	~	P	4 MONTHS	P	4 YEARS	NIL	B/L SINONASAL POLYPOSIS	4	4	2	2	FESS-B/L MMA,AE,PE	76	114	76	89	L	23	R	20	Nil
586394	28	F	COOLIE	P	8 MONTHS	A	~	P	2 MONTHS	A	~	NIL	B/L SINONASAL POLYPOSIS	5	4	3	3	FESS-B/L MMA,AE,PE	82	122	70	87	L	20	R	24	Nil
585421	58	M	FARMER	P	1 YEAR	A	~	P	3 MONTHS	P	2 YEARS	NIL	B/L SINONASAL POLYPOSIS	4	5	3	3	FESS-B/L MMA,AE,PE	80	116	78	91	L	18	R	22	Nil
490749	38	F	FARMER	P	3 YEAR	P	3 MONTH	P	5 MONTHS	A	~	NIL	CHRONIC RHINOSINUSITIS+DNS	9	6	3	3	FESS-B/L MMA,AE,PE,S,FRC+SEPTO	78	118	70	86	R	23	L	20	Nil
568075	60	M	FARMER	P	2 YEARS	A	~	A	~	P	3 YEARS	NIL	B/L SINONASAL POLYPOSIS	5	4	2	2	FESS-B/L MMA,AE,PE	84	120	74	89	L	24	R	20	Nil
609725	25	M	COOLIE	P	2.5 YEARS	P	3 MONTHS	P	3 MONTHS	A	~	NIL	B/L SINONASAL POLYPOSIS	5	4	4	3	FESS-B/L MMA,AE,PE	76	122	80	94	L	18	R	23	Nil
471103	24	F	HOMEMAKER	P	2 MONTHS	P	3 MONTHS	P	6 MONTHS	P	2 YEARS	NIL	ALLERGIC RHINITIS	~	~	~	~	B/L IT	80	116	70	85	R	20	L	23	Nil
568257	27	F	HOMEMAKER	A	~	P	6 MONTHS	A	~	P	5 YEARS	NIL	ALLERGIC RHINITIS	~	~	~	~	B/L IT	75	118	72	87	R	22	L	18	Nil
499940	32	F	CLERK	A	~	A	~	A	~	P	3 YEARS	NIL	ALLERGIC RHINITIS	~	~	~	~	B/L IT	81	122	80	94	R	21	L	17	Nil
609728	45	M	FARMER	P	1 YEAR	P	2 MONTHS	A	~	A	~	NIL	B/L SINONASAL POLYPOSIS	3	3	4	4	FESS-B/L MMA,AE,PE	77	120	70	87	L	20	R	20	Nil
597295	22	M	STUDENT	A	~	P	2 MONTHS	P	3 MONTHS	A	~	NIL	CHRONIC MAXILLARY SINUSITIS	5	4	3	2	FESS-B/L MMA	80	116	76	89	L	17	R	22	Nil
442618	78	M	FARMER	P	8 MONTHS	P	3 MONTHS	A	~	A	~	NIL	B/L SINONASAL POLYPOSIS	4	6	3	4	FESS-B/L MMA,AE,PE	82	118	78	91	L	23	R	18	Nil
596308	43	M	COOLIE	A	~	P	6 MONTHS	P	6 MONTHS	A	~	NIL	CHRONIC RHINOSINUSITIS	7	6	4	3	FESS-B/L MMA,AE,PE,S,FRC	78	114	80	91	R	18	L	18	Nil
650424	40	F	HOMEMAKER	P	1 YEAR	P	4 MONTHS	A	~	P	4 YEARS	NIL	B/L SINONASAL POLYPOSIS	5	3	2	3	FESS-B/L MMA,AE,PE	84	120	72	88	R	22	L	17	Nil
655608	44	M	COOLIE	P	4 YEARS	P	3 YEARS	A	~	P	3 YEARS	NIL	ALLERGIC RHINITIS WITH DNS	~	~	~	~	SEPTO+B/L IT	76	116	78	91	R	21	L	15	Nil
639641	26	F	HOMEMAKER	A	~	P	1 YEAR	A	~	P	2 YEARS	NIL	ALLERGIC RHINITIS	~	~	~	~	B/L IT	80	122	70	87	L	20	R	16	Nil
557687	48	F	FARMER	A	~	P	6 MONTHS	P	4 MONTHS	A	~	NIL	CHRONIC MAXILLARY SINUSITIS	4	4	3	3	FESS-B/L MMA	83	120	78	92	R	20	L	20	Nil
663276	60	F	FARMER	P	1 YEAR	A	~	A	~	A	~	NIL	B/L SINONASAL POLYPOSIS	5	5	3	3	B/L MMA,AE,PE	78	118	76	90	R	17	L	22	Nil
499940	35	F	CLERK	A	~	A	~	A	~	P	4 YEARS	NIL	ALLERGIC RHINITIS	~	~	~	~	B/L IT	77	114	80	91	L	20	R	20	Nil
425833	60	M	FARMER	P	3 YEARS	P	3 MONTHS	A	~	P	2 YEARS	NIL	B/L SINONASAL POLYPOSIS	4	5	2	2	FESS-B/L MMA,AE,PE	82	122	74	90	L	18	R	23	Nil
438962	32	F	HOMEMAKER	P	6 MONTHS	A	~	A	~	A	~	NIL	B/L SINONASAL POLYPOSIS	4	3	3	2	FESS-B/L MMA,AE,PE	75	118	78	91	R	20	L	17	Nil
484444	35	F	FARMER	A	~	P	1.5 YEARS	A	~	P	3 YEARS	NIL	ALLERGIC RHINITIS	~	~	~	~	B/L IT	84	120	70	87	R	20	L	20	Nil
497793	36	F	HOMEMAKER	A	~	P	3 MONTHS	P	6 MONTHS	A	~	NIL	CHRONIC RHINOSINUSITIS	8	8	3	2	FESS-B/L MMA,AE,PE,S,FRC	86	114	78	90	L	22	R	20	Nil
616009	62	M	FARMER	P	2 MONTHS	P	6 MONTHS	P	3 MONTHS	A	~	NIL	CHRONIC RHINOSINUSITIS	8	7	2	3	FESS-B/L MMA,AE,PE,S,FRC	76	120	70	87	L	20	R	20	Nil
535643	57	M	COOLIE	P	6 MONTHS	A	~	P	3 MONTHS	A	~	NIL	B/L SINONASAL POLYPOSIS	5	6	2	2	FESS-B/L MMA,AE,PE	80	122	80	94	L	18	R	22	Nil
608511	29	M	CLERK	A	~	P	6 MONTHS	A	~	P	2 YEARS	NIL	ALLERGIC RHINITIS+DNS	~	~	~	~	B/L IT+SEPTO	82	118	78	91	R	20	L	16	Nil
612380	18	M	STUDENT	A	~	P	1 YEAR	A	~	P	4 YEARS	NIL	ALLERGIC RHINITIS+DNS	~	~	~	~	B/L IT+SEPTO	78	116	80	92	L	20	R	14	Nil
642048	75	M	FARMER	A	~	P	2 MONTHS	A	~	A	~	NIL	B/L MAXILLARY SINUSITIS	4	4	4	3	FESS-B/L MMA	80	120	70	87	R	20	L	20	Nil

644039	32	F	HOMEMAKER	P	4 MONTHS	P	4 MONTHS	A	~	A	~	NIL	B/L MAXILLARY SINUSITIS+DNS	4	3	3	4	FESS-B/L MMA+SEPTO	83	120	76	91	R	20	L	22	Nil
664496	19	M	STUDENT	P	2 MONTHS	P	1 YEAR	A	~	P	3 YEARS	NIL	ALLERGIC RHINITIS+DNS	~	~	~	~	B/L IT+SEPTO	76	116	80	92	L	22	R	18	Nil
651927	29	M	COOLIE	P	1 YEAR	A	~	A	~	P	2 YEARS	NIL	B/L SINONASAL POLYPOSIS	5	6	2	2	FESS-B/L MMA,AE,PE	82	122	72	89	R	22	L	16	Nil
686894	59	F	COOLIE	A	~	P	2 MONTHS	P	7 MONTHS	A	~	NIL	CHRONIC RHINOSINUSITIS	6	7	4	3	FESS-B/L MMA,AE,PE,S,FRC	77	118	70	86	R	21	L	16	Nil
684420	72	M	FARMER	P	1.5 YEARS	P	2 MONTHS	P	2 MONTHS	P	2 YEARS	NIL	B/L SINONASAL POLYPOSIS	6	6	3	3	FESS-B/L MMA,AE,PE+SEPTO	75	118	78	91	R	20	L	20	Nil
691268	45	F	HOMEMAKER	P	2 YEARS	P	3 MONTHS	P	6 MONTHS	A	~	NIL	CHRONIC RHINOSINUSITIS+DNS	6	5	3	3	FESS-B/L MMA,AE,PE+SEPTO	83	122	70	87	L	22	R	18	Nil
679414	29	M	COOLIE	A	~	P	1 YEAR	A	~	P	3 YEARS	NIL	ALLERGIC RHINITIS+DNS	~	~	~	~	B/L IT+SEPTO	78	120	80	93	L	20	R	20	Nil
622008	22	F	HOMEMAKER	A	~	P	1 YEAR	P	~	P	4 YEARS	NIL	ALLERGIC RHINITIS+DNS	~	~	~	~	B/L IT+SEPTO	81	118	76	90	L	18	R	22	Nil
692864	40	F	HOMEMAKER	A	~	P	2 MONTHS	P	3 MONTHS	P	2 YEARS	NIL	CHRONIC RHINOSINUSITIS	6	7	4	3	FESS-B/L MMA,AE,PE,S,FRC + SEPTO	84	116	78	91	L	18	R	22	Nil
706003	36	F	FARMER	P	9 MONTHS	A	~	A	~	A	~	NIL	B/L SINONASAL POLYPOSIS	5	6	2	2	FESS-B/L MMA,AE,PE	78	118	80	93	L	22	R	18	Nil
733945	36	F	COOLIE	P	1 YEAR	A	~	P	2 MONTHS	A	~	NIL	B/L SINONASAL POLYPOSIS	5	5	3	2	FESS-B/L MMA,AE,PE	80	120	80	93	R	16	L	22	Nil
727658	19	M	STUDENT	P	3 YEARS	P	3 MONTHS	P	5 MONTHS	A	~	NIL	CHRONIC RHINOSINUSITIS+DNS	7	7	3	2	FESS-B/L MMA,AE,PE,S,FRC+ SEPTO+B/L IT	82	122	70	87	R	20	L	20	Nil
728308	30	M	COOLIE	P	6 MONTHS	A	~	A	~	A	~	NIL	B/L SINONASAL POLYPOSIS	3	4	2	2	FESS-B/L MMA,AE,PE	76	118	80	93	L	22	R	18	Nil
684656	26	F	HOMEMAKER	P	4 MONTHS	P	6 MONTHS	A	~	P	2 YEARS	NIL	B/L SINONASAL POLYPOSIS+DNS+B/L ITH	5	5	3	2	FESS-B/L MMA,AE,PE,S,FRC+ SEPTO+B/L IT	84	116	72	87	R	20	L	15	Nil
724679	68	F	FARMER	P	2 YEARS	P	3 MONTHS	P	8 MONTHS	A	~	NIL	CHRONIC RHINOSINUSITIS+DNS	7	5	4	4	FESS-B/L MMA,AE,PE,S,FRC+ SEPTO	81	118	78	91	L	22	R	16	Nil
743658	50	F	FARMER	P	1 YEAR	P	2 MONTHS	A	~	P	2 YEARS	NIL	B/L SINONASAL POLYPOSIS	6	5	3	3	FESS-B/L MMA,AE,PE	77	122	70	87	R	20	R	20	Nil
765197	38	M	COOLIE	P	1 YEAR	A	~	A	~	A	~	NIL	B/L SINONASAL POLYPOSIS	4	3	2	2	FESS-B/L MMA,AE,PE	80	120	78	92	R	15	L	20	Nil
769421	30	M	CLERK	P	6 MONTHS	P	5 MONTHS	P	3 MONTHS	P	2 YEARS	NIL	B/L SINONASAL POLYPOSIS+DNS	7	5	3	3	FESS-B/L MMA,AE,PE+SEPTO	82	118	80	93	L	20	R	20	Nil
521700	45	M	CLERK	P	8 MONTHS	P	4 MONTHS	A	~	P	3 YEARS	NIL	B/L SINONASAL POLYPOSIS	6	6	4	3	FESS-B/L MMA,AE,PE	80	120	76	91	R	14	L	20	Nil
773808	49	M	FARMER	P	1 YEAR	A	~	P	2 MONTHS	A	~	NIL	B/L SINONASAL POLYP+DNS	4	4	2	2	FESS-B/L MMA,AE,PE+SEPTO	79	122	70	87	L	16	R	20	Nil